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Excursions
into
Surgical Subjects

By

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PREFACE

The first five Excursions in the present volume represent, in substance, a series of lectures delivered by the senior author in July, 1922, at Washington University, Seattle, Wash., in the Extension Course for Graduate Physicians of that institution. It was a very great privilege to address this body of eager and enthusiastic fellow-members of our great profession, and it is gratifying to have this opportunity of acknowledging the pleasure and satisfaction derived from that experience.

The pathologic and physiologic phases in these lectures have been developed from several sources, the literature, our experiences, discussions in our staff meetings, and odds and ends of experimental work, coupled with observations in our follow-up clinic. Some of the subject matter has already appeared in scattered publications.

Of the other three essays in the volume the one on Living Pathology has already appeared in print in the Journal of the American Medical Association, to which due credit is hereby acknowledged. The other two are now presented to a larger public for the first time. All three, we believe, are timely subjects which bear repeated emphasis and discussion.

It is the earnest wish of the authors that these Excursions may stimulate the traveler in the world of surgery to continued inquiry and investigation in the by-ways of surgery and thus open up and develop new paths which may perhaps become landmarks to future travelers.

The authors desire to record their grateful acknowledgment to Dr. I. S. Ravdin and to Miss A. M. Jastrow for assistance rendered in the preparation of the volume; also to Mr. Irwin Faber for painstaking and satisfactory drawings. To their publishers they desire to express their appreciation for the kind consideration shown them at all times.

JOHN B. DEAVER.
STANLEY P. REIMANN.

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CONTENTS

PAGE

PEPTIC ULCER.. 1

JAUNDICE.. 53

DISEASES OF THE BILE-PASSAGES........................ 67

TRIALS, TRIBULATIONS, AND JOYS OF A SURGEON........... 101

SOME SURGICAL CONDITIONS OF THE INTESTINAL TRACT...... 117

THE CONTRIBUTION OF PASTEUR TO MODERN SURGERY...... 149

MEDICAL EDUCATION AND EDUCATORS..................... 161

LIVING PATHOLOGY................................. 169

INDEX.. 183

EXCURSIONS INTO SURGICAL SUBJECTS

PEPTIC ULCER

For the first of this series of lectures I have chosen the subject of peptic ulcer because it seems to be continually growing in interest and importance as one of the most frequent benign lesions of the upper gastro-intestinal tract. It is of like interest to the clinician, the surgeon, the bacteriologist, the physiologist, and the pathologist. The term "peptic ulcer," an ulcer occurring in the portion of the intestinal tract in which hydrochloric acid is normally present, is comprehensive, inasmuch as it includes primary ulceration of the stomach and the duodenum as well as the occasional marginal or gastrojejunal and the jejunal ulcer. Cruveilhier is usually given the credit for having (in 1835) published the first clinical and anatomic description of peptic ulcer, although Matthew Baillie in 1793 had already given an accurate anatomic description of ulcer of the stomach. Ulceration of the stomach, however, has been known from the time of Hippocrates, who, from the appearance of the vomitus and the stools, named the condition "morbus niger," but he probably had little idea of the actual lesion.

It was Frederich Hoffmann, of Halle (1660–1742), who recognized that the vomitus consisted largely of blood coming from the vessels of the stomach wall, which, in his opinion, were ulcerated by the action of the acid and acrid gastric juices. Johann Peter Frank (1745–1821) was the first to employ the term "ulcus ventriculi," and it is interesting to note that, although surgery for this condition was then unthought of, he employed the principles of treatment which both the clinician and the surgeon use today—absolute rest, "summa quis corporis imperanda," the application of snow to the surface of the abdomen, a milk diet, and the internal use of a styptic agent. Archibald Mallock, in a very interesting paper entitled An Early Record of Perforating Duodenal Ulcer, has shown that Jacopa Penada in 1791 gave a detailed description, accompanied by drawings, of the autopsy findings of a case of perforating duodenal ulcer; later, in 1824, Abercrombie also described the lesion. But the history of the surgery of peptic ulcer begins at a much later date. In 1881 Rydygier resected an ulcer from the posterior wall of the stomach and cured his patient. In 1893 Doyen performed a gastrojejunostomy for the treatment of an ulcer, and in the same year Braun performed the same operation for a perforating ulcer. The year 1893 was important also because of the comprehensive studies by Perry and Shaw on diseases of the duodenum, which aroused the interest of surgeons who were then beginning to realize that perforating ulcers were within their province. In that year also Codvilla, of Italy, performed an operation for chronic duodenal ulcer. The names of Mayo-Robson, Kriege, Mikulicz, Krause, Czerny, Moynihan, Crile, and the Mayos all deserve to be mentioned as pioneers in this field of surgery.

The frequency of the chronic ulcer was first emphasized by Robert Weir in 1900, and in 1903 there appeared Moynihan's first work on the surgical treatment of gastric and duodenal ulcer, followed several years later by his classic study of duodenal ulcer with its graphic description of the clinical picture of the disease and which, to a great extent, established the differentiation between the gastric and duodenal location of the lesion.

The dividing line between gastric and duodenal ulcer is now generally accepted to be the pyloric vein, or veins. This vein, known as the anterior pyloric vein of Mayo, passes from below the superior to the inferior border of the pylorus opposite its junction with the duodenum, and is the visible guide to the position of the pyloric ring. Although as constant a landmark as a vein can be, it is not the most essential mark; the actual position of the pylorus can well be seen as a thin white line which, as has been pointed out by Moynihan, becomes clearer when the pylorus is held forward. It should, however, be remembered that ulcers rarely begin at the pylorus itself; they usually originate on the duodenal side and extend upward into the gastric mucosa.

The essential condition for the development of ulceration of the stomach or duodenum is erosion of the mucosa, based on impaired resistance of the latter. What leads to the primary erosion is still a matter of controversy. It has been variously attributed to bacterial infection, burns, venous congestion, pressure necrosis, emboli, toxins, and many other conditions. C. H. Mayo, in discussing the etiology, says: "I believe that peptic ulcer is undoubtedly developed by a combination of local chemical effects, possibly causing prolonged vessel spasm or claudication, a counterpart of Raynaud's disease or scleroderma, or by direct interference with the circulation through infarction by emboli of bacteria, chemically and mechanically active in the tissues, and thereby lowering the local resistance to the action of digestive fluids."

In plain clinical language this can be expressed by one word—infection—which originates from a focus within the body. Ever since Rosenow's investigations into the selective affinity of certain organisms for certain parts of the body, the appendix vermiformis is considered as probably the most potent source of focal infection which gives rise to gastric and duodenal ulcer. I believe it is Moynihan himself who says the origin of numerous gastric and duodenal ulcers is to be found in the appendix.

The tendency to infection in the gastro-intestinal tract is ably demonstrated by Bolton's histologic studies. He describes the sequence of events in the development of gastric ulcer, as localized necrosis of the mucous membrane due not to the food, but to bacteria or their toxins; localized hemorrhage and inflammation in the lymphatic follicles at the bases of the glands upon the muscularis mucosa, these follicles being more numerous along the lesser curvature and especially toward the pylorus. A submucous abscess arising in some of these follicles and rupturing into the cavity of the stomach permits the gastric juice to act on the base of the ulceration thus exposed.

Gastric ulcers are frequently classified as acute and chronic, and there is strong reason to believe that every chronic ulcer was at one time an acute ulcer, which has failed to heal. The so-called acute or early lesion is generally present as an erosion or a mere abrasion of the gastric mucosa, more or less superficial, and causing no symptoms and generally healing without leaving a scar. Ulceration, as distinguished from erosion, extends deeper into the gastric wall, and frequently gives rise to serious hemorrhage and possibly also to perforation. When located at the cardiac half of the stomach such an ulcerated area is more apt to heal than when it develops near the pylorus. This may be due to the "law of localization of diseased action," that is, the parts enjoying the most rest being less liable to involvement by structural disease. It is when this type of ulceration fails to heal that it merges into the true round or punched-out gastric lesion. As this type of ulcer progresses it assumes a conical shape with sloping edges and thickened margins; the surrounding mucosa becomes puckered, gradually the edges are undermined, the submucosa becomes thickened, followed by formation of sloughs which show evidence of the action of the acid gastric juice; finally there results the chronic, callous, indurated ulcer, with its thick, hard, elevated margins and its base covered with tenacious mucus. When this stage is reached a permanent chronicity is established—"once a chronic ulcer always a chronic ulcer." While early erosions and ulcerations in the stomach wall may and often do heal spontaneously, it is readily seen that when healing does not take place the disease can scarcely progress without causing more or less marked symptoms. If the early symptoms are such as to command attention, a cure by medical treatment may be possible. But it is doubtful whether a true gastric ulcer as distinguished from an erosion can ever actually be healed by medication. Latency and a period of relief may be obtained, but recurrence of symptoms is practically inevitable. Indeed, I cannot help thinking that the claims with regard to the healing of acute ulcers are based more upon faith than upon fact. There is, as far as I know, no definite way of telling when or whether an acute ulcer is healed, or when it becomes chronic. All that I know is what I have learned from my operative experience, that the crippling attacks of upper abdominal distress are the result of the chronic ulcer. The few cases of acute ulcer which I have seen are just sufficient to whet my curiosity, but not enough to change my opinion.

GASTRIC ULCER

Gastric ulcer is a disease of middle life, and shows a marked preference for the male sex, the proportion being about 3 to 1. Its chief characteristic is the periodicity of attacks, and its outstanding symptom, epigastric pain after meals. The attacks may recur two or three times a year, extending over several weeks, with complete freedom from symptoms in the intervals. It is this latter fact which often leads the medical man to think he has obtained a cure in a given case.

Regularity in the time of the onset of pain is another prominent symptom of gastric ulcer. It seems to occur with clock-like regularity, the interval between eating and the onset of pain being fairly constant, from one-half to two hours after a meal. The time relation between the the meal and the onset of pain may sometimes indicate the location of the ulcer; that is to say, if the pain comes on immediately or within about half an hour after eating the ulcer is apt to be located near the esophagus, while if the pain is delayed from one to two hours, it is probably prepyloric; if the pain comes on still later, the lesion is probably beyond the pylorus.

Relief of pain by taking food or alkalies may be said to be characteristic of gastric ulcer, that is, as long as stenosis has not developed or adhesions have not formed. The pain in these circumstances is apt to change its character and, in the case of gastric ulcer, to be longer delayed. As a rule, the pain is described as a dull gnawing ache in the epigastrium, more frequently to the left of the midline, often extending through to the back, and always occurring at the same site. It may also be influenced by the type of food, being aggravated by coarse solid food, by large amounts of food, as well as by foods that produce a free flow of gastric juice. It is characteristic also of gastric ulcer that the pain which comes on after meals gradually subsides before the next meal is taken. In duodenal ulcer, on the other hand, the pain persists until relieved by food. Moynihan happily expresses this as a quadruple rhythm in gastric ulcer: food, comfort, pain, comfort; and a triple rhythm in duodenal ulcer: food, comfort, pain.

Vomiting is not a very frequent symptom; oftentimes it is self-induced in the desire to relieve the pain; but when spontaneous it occurs from one-half to one hour after eating, the vomitus usually being acid, malodorous, and sometimes streaked with blood. Hematemesis, however, is not very frequent. It may occur at any time that the ulcer invades a blood-vessel; or it may result from a sudden increase in arterial pressure. It must be borne in mind, however, that hemorrhage also occurs with other conditions, such as cirrhosis of the liver, splenic anemia, and occasionally from toxic conditions resulting from disease of the gall-bladder and of the appendix. Sometimes hemorrhage into the stomach takes place without causing vomiting; the blood remaining in the stomach may become mixed with the food subsequently taken, then passing out with the stools. Secondary anemia is usually present for a few days after a hemorrhage, manifested by a marked decrease in hemoglobin and a less marked decrease in red blood-cells. Irregular fever is also sometimes observed.

A careful history in cases of suspected ulcer is of primary importance, inasmuch as the greatest reliance can be placed upon the pain and its characteristics and on the periodicity of the attacks. Clinically there are various means of confirming the suspicion of the presence of ulcer. Physical examination is important in spite of all our diagnostic aids. Careful palpation will reveal an area of tenderness, which, if the ulcer is on the lesser curvature, is usually high up along the left costal margin; in prepyloric ulcer the area of tenderness, of course, is lower down and more median. While in the presence of actual deformity, stenosis, or obstruction, fluoroscopic examination may indicate the presence of ulcer, it cannot be said to do so in all conditions. Personally, I place greater reliance on the results of a gastric analysis in order to ascertain the degree of retention, acidity, etc. The capacity of the stomach can be determined by inflating it with air and percussing its outlines, and marking them on the abdominal wall. After allowing the air to escape, the stomach can then be filled with water, and again, by percussion, the area of dulness can be ascertained; this gives information which cannot be obtained in any other way. If this area corresponds with the area of resonance we may be reasonably sure of our findings. If the *x*-ray were more reliable than it is, I naturally would prefer it because it is so much easier for the patient. But I have so often had *x*-ray reports indicate retention in the absence of pyloric obstruction, while with the stomach-tube I have been able to satisfy myself that there was no retention, and have had the fact confirmed at operation, that I am rather conservative in depending on radiographic reports. I have little to say with regard to acidity as a clinical symptom of gastric ulcer because of the varying opinions as to what is considered the normal flow of hydrochloric acid. We all know that it is influenced by the time of day, the character of the food, and also by the nervous state of the individual. Variations of opinion also exist as to whether hyperacidity or hypo-acidity is an accompaniment of gastric ulcer, since ulcers not infrequently progress in the presence of normal acidity. Nevertheless, it remains a fact that ulceration of the stomach leads to physiologic disturbances in motor and secretory function, which should be studied in each individual case in order to obtain the best results from treatment by the comparison of function before and after treatment.

Differential diagnosis is important because other gastric disturbances also present the ulcer syndrome. Gastric neurosis with spasm and hyperchlorhydria, though not so frequent a diagnosis as formerly, is nevertheles a distinct entity, but not a surgical condition. The differential diagnosis between gastric neurosis and ulcer is often difficult. I have frequently operated upon patients who for years had been treated for a neurosis, when an ulcer was the cause of all the symptoms. In neurosis the important features are the irregular intervals between attacks, their independence of diet and the anomalies referable to a nervous state, which are so frequently present. Disease of the gall-bladder and of the appendix causing reflex pylorospasm may also present the clinical symptoms of ulcer.

Hemorrhage, while it is said to occur in about 25 per cent. of ulcer cases, frequently accompanies other upper abdominal conditions, such as disease of the liver, the spleen, and the pancreas. Moynihan, in calling attention to some of the factors causing disappointments after gastro-enterostomy, assigns an important place to incorrect diagnosis and injudicious or unnecessary operations. Among conditions in which a mistaken diagnosis is often made are tabes dorsalis, chronic appendicitis, tuberculosis of the intestines, disease of the gallbladder, cirrhosis of the liver with hemorrhage, splenic anemia, the vomiting of pregnancy, ptosis of the kidney and of the stomach, etc.

If the diagnosis of chronic gastric ulcer could always be accurately made we might say that the numerous reported cures by non-surgical means were actual ones. I hope I have made it clear that there is no absolute certainty that the ulcer was present in these cases. In fact I might say of gastric ulcer that the certainty is not even reasonable. A favorite argument in support of the medical treatment of peptic ulcer is the fact that so many patients have a very long previous history of digestive disturbances, meaning that the disease is not dangerous to life. This argument, however, does not take into consideration the economic factors of expense and disability, and the emphasis placed by modern medicine upon the economic and social aspects of disease. Moreover, even if they were genuine cases, there are few, very few individuals so situated financially and socially as to be able to undergo the prolonged careful supervision, to say nothing of having the intelligence required to carry out the régime of medical treatment. It is for this reason that I say that for chronic gastric ulcer, as distinguished from all early erosions, the treatment is eminently surgical.

Of the various methods of medical treatment that of Sippy, in changing the environment of the ulcer and giving it a chance to heal, seems the most logical. But it must be borne in mind that the results of gastric analysis show that alkalies are indicated in scarcely fifty per cent. of the cases; and in fact, that a considerable number indicate acid medication. In the long run, too, gastrojejunostomy with excision of the ulcer provides a greater opportunity for cure and offers a longer expectancy of life.

In operating for the relief of gastric ulcer, when the ulcer is small and has not seriously affected the gastric mechanism, simple excision and closing of the gastrotomy wound may suffice. But this can rarely be done, because nearly all ulcers which come to operation are old and more or less calloused, too often due to prolonged, and I need scarcely add, unsuccessful, medical treatment. These large ulcers demand more extensive surgery. On the whole, excision followed by gastro-enterostomy has proved satisfactory both as to immediate and end-results, but with the advance in surgical technic the more radical operations, such as pylorectomy, subtotal gastrectomy, and transgastric resection, are rapidly gaining favor both here and abroad. If the theory of the neutralization of the gastric contents by the reflux of intestinal and pancreatic secretions, and of the limitation of these, so that they do not reach all parts of the stomach alike, is correct, we have a strong argument in favor of radical operation for gastric ulcer. There are, however, other more potent reasons for the radical treatment. The disease is a much more serious one than ulcer of the duodenum, and, furthermore, it carries with it the possibility of carcinomatous transformation. Whatever may be the percentage of this change, it is one that must be reckoned with.

Before entering upon the discussion of the technic of the more radical operations on the stomach, I should like to say a few words about pyloroplasty, a method originated, as you know, by Finney, of Baltimore, and one which has given him such good results. Finney and the other advocates of his method claim that its effects are the same as the most radical operation. In other words, the ulceration being the cause and not the result of hyperacidity and hypersecretion, excision of the ulcer removes the local condition, while pyloroplasty promotes rapid evacuation of the stomach contents, thus preventing hypersecretion, and, moreover, leaves the normal relations of the stomach and the duodenum intact. The conservative nature of the operation, of course, is greatly in its favor, and, taking an ulcer diathesis for granted (that is to say, hyperacidity once established is likely to continue or to recur), it is a rational method. But again it must be borne in mind that only about 50 per cent. of cases of gastric and duodenal ulcer present hyperacidity before operation. Furthermore, Finney claims that the operation is simple and less time consuming than gastro-enterostomy, and therefore safer. While this may be the case with Finney himself, I believe it is not the experience of many other surgeons. Personally, I think the method may be indicated in the absence of dilatation, that is, where the stomach has lost its power of contraction; and in the absence of perigastric adhesions and when the pylorus is not involved in cicatricial tissue. We all know that adhesions are one of nature's safeguards which she sets up on every occasion and which should be treated with respect. In the choice of an operation the time and the place are important, but of greatest importance is the proper selection of the case. It seems, however, that pyloroplasty with excision of the ulcer, when possible, is again gaining favor.

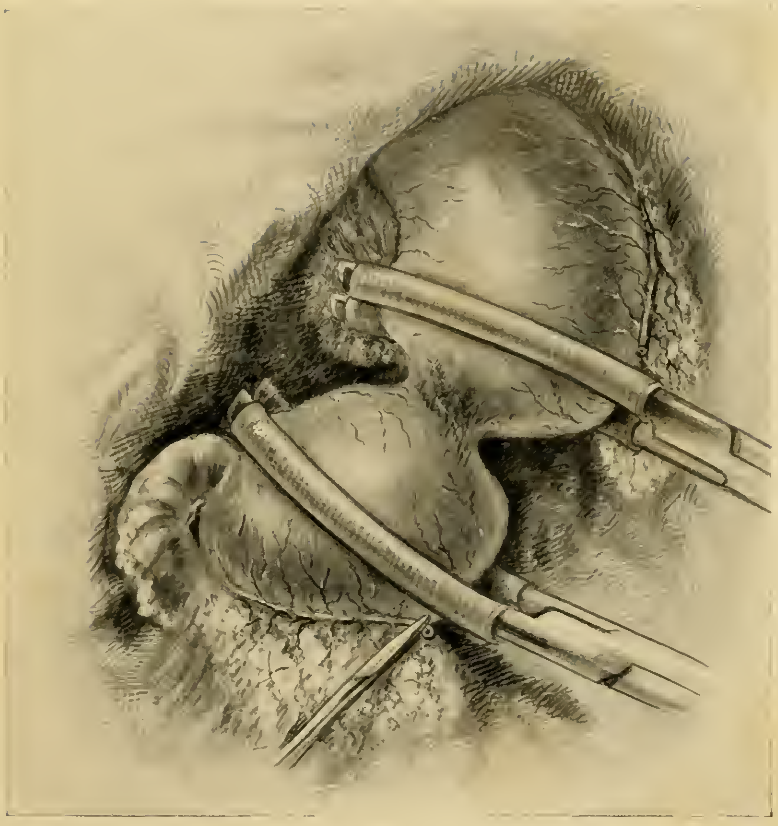

Fig. 1.—The clamps applied preparatory to a circular resection of the stomach.

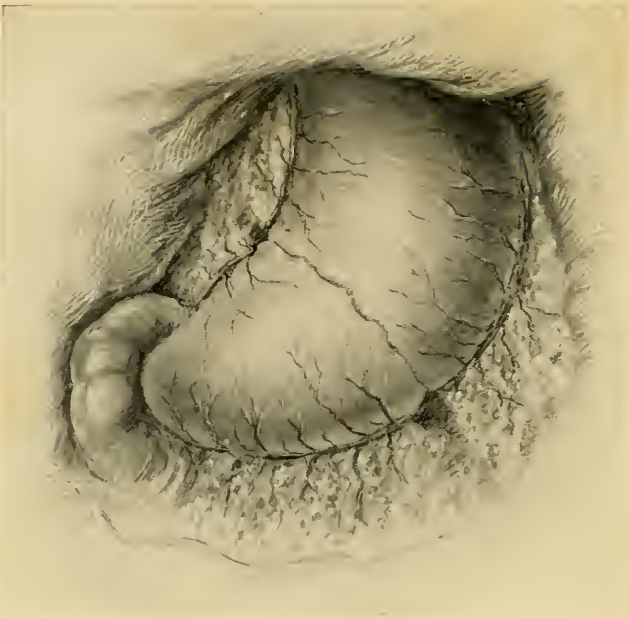

Fig. 2.—The operation of circular resection completed by the union of the remaining two parts of the stomach.

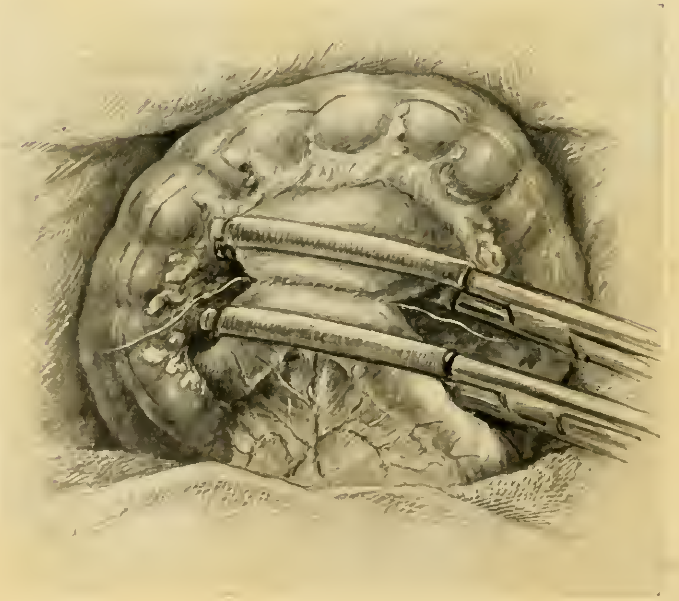

Fig. 3.—The gastro-enterostomy practically completed.

For ulcers near the pylorus, whether gastric or duodenal in origin, I am in the habit of doing a pylorectomy whenever I can; that is, I remove the pylorus with its antrum, including, of course, the ulcer-bearing area, terminating the operation with a posterior gastro-enterostomy, or performing the typical Polya operation. An ulcer located not far from the pylorus allows excision of the pylorus with the portion of the stomach involved and the making of a gastroduodenostomy, or, if you like, a pyloroplasty.

For ulcers proximal to the pylorus, near, or on the lesser curvature of the stomach a partial gastrectomy, which includes the lesser curvature, is in order, while if the greater curvature is also involved, a subtotal gastrectomy is indicated. Circular or sleeve resection is the method I employ for ulcers located approximately in the middle portion of the stomach, especially the saddle-back ulcers with hour-glass constriction. Considering the extent and the gravity of these procedures, and the usually greater debilitating effects of gastric compared with duodenal ulcer, the operation is a comparatively safe one.

The end-results of radical operations are more than satisfactory. The Follow-up Department of the Lankenau Hospital (organized in October, 1920) has been able to trace 50 patients who were operated upon for gastric or duodenal ulcer between the years 1909–1920. The reported cures in that time average about 94 per cent. Of these, 12 per cent. had been subjected to radical operation consisting of either a pylorectomy or a sleeve resection, or a subtotal gastrectomy. One of the latter had been done in 1910 and the patient is today in perfect health.

Duodenal Ulcer

The outstanding characteristics which distinguish a duodenal from a gastric ulcer are its greater frequency and its lesser tendency to malignant transformation. The proportion of duodenal to gastric ulcer is generally estimated as 4 to 1; cancer of the duodenum is very rare. In a recent investigation of the latter question conducted by Dr. Ravdin and myself, it was found that among 151,201 autopsy records collected by Ravdin, carcinoma of the duodenum was mentioned only 50 times (0.033 per cent.). While, therefore, the bearer of duodenal ulcer is only exceptionally threatened with this menace, he is confronted with the danger of perforation and hemorrhage; perforation is generally estimated to occur nearly three times as frequently in duodenal as in gastric ulcer.

Duodenal ulcer resembles the gastric lesion in its predilection for the male sex and its occurrence mainly between the ages of thirty and fifty years. A word as to multiple ulcers. In his earliest study of peptic ulcer Moynihan made the statement that a duodenal ulcer seldom exists without "unmistakable evidence of gastric ulcer," but subsequent observations failed to substantiate this statement. Bolton claims that from 10 to 20 per cent. of chronic gastric ulcers are multiple. I cannot confirm this statement. As to multiple duodenal ulcers, I find that in my recent records (Lankenau Hospital), about 3 per cent. of the cases presented two ulcers, one in the first portion and one in the second portion of the duodenum. These same records also show that 23 per cent. of the single ulcers are located near the pylorus, 46 per cent. in the first part of the duodenum, that is, between the entrance of the common duct and the pylorus; 28 per cent. in the second part of the duodenum; the remaining 3 per cent. being the multiple ulcers already mentioned. At the Mayo Clinic in 5723 operations for gastric or duodenal ulcers both gastric and duodenal lesions were found in the same patient in only 203 instances (3.5 per cent.).

The frequency of the first part of the duodenum as the site of ulcer is variously explained, the most plausible reason being that the acid chyme, still containing active pepsin, remains in this area until it is neutralized by the alkaline secretion. Another factor of importance has been brought out by Wilkie, who discovered that a single vessel supplies the first 1½ inches of the duodenum with an apparently inadequate nutrition, while Reeves and others have shown that the anatomic arrangement of the arterioles gives an opportunity for stasis, thus creating a predisposition to thrombosis.

The clinical history of duodenal ulcer, like that of gastric ulcer, is characterized by long-standing "stomach trouble," periodicity of attacks, and complete or almost complete well-being between attacks. Latency is more marked in duodenal than in gastric ulcer, at least 75 per cent. of the cases giving a previous history of digestive disturbances. The pain of duodenal ulcer occurs late, that is, three or four hours after eating. Hunger-pain at night is also more frequent in duodenal ulcer. As already mentioned the pain persists until relieved by food or by alkalies.

Vomiting is rare, as are also hematemesis and melena. Jaundice may be present in the cases in which the papilla of Vater is invaded by the ulcer, but if present it is more likely due to concomitant disease of the biliary passages.

Physical examination usually reveals more or less tenderness near the right costal margin and extending through the descending portion of the duodenum. x-Ray study at the hands of the expert and with the taking of a great number of plates is useful in confirming the clinical diagnosis of duodenal ulcer. But in the hands of the average roentgenologist and where only one or two, or perhaps three, different exposures are made, x-ray diagnosis, except in the presence of deformity due to roughening of the mucosa or to crippling adhesions, is, I repeat, not altogether reliable.

In analyzing gastric contents acidity in duodenal ulcer varies almost as much as in gastric ulcer, and therefore offers no definite conclusions. But the presence of occult blood in the gastric contents or in the stools is of some diagnostic value.

The main point after all in the diagnosis of duodenal ulcer is the history of the case. The fact that the "typical" history is so much more frequent than in gastric ulcer, makes the diagnosis of the duodenal lesion comparatively easy; differential diagnosis, however, is oftentimes called for. For differentiating duodenal from gastric ulceration we have four distinguishing features: greater frequency and latency, delayed pain after meals, and the prompt relief following food or an alkali. The most common error in the diagnosis of duodenal ulcer is probably that of mistaking it for chronic appendicitis. The differentiation is not always easy, particularly as we are so accustomed to having appendicitis assume the picture of any of the more common disorders of the upper abdomen. The symptoms of chronic appendicitis, however, usually do not present the same relation to food, either in time, or type, or relief; while the intervals between attacks are not apt to be so long nor so free from digestive disturbances. In suspected cases of appendicitis, where the condition of the appendix at operation does not seem to be sufficient to have caused the symptoms, it is well to look for duodenal ulcer. I might also say, vice versa, were it not my practice, circumstances permitting, to remove the appendix in all cases of chronic duodenal ulcer as well as in all abdominal operations. A third condition which is often confused with duodenal ulcer is disease of the gall-bladder. The symptoms of the two disorders are frequently very much alike. Even jaundice and the pain referred to the right side and between the shoulder blades, so characteristic of cholecystitis, may be present in duodenal ulcer. But, on the other hand, the disease of the gall-bladder rarely presents the same periodic and prolonged attacks of pain definitely related to food intake. The simultaneous existence of the two diseases should, however, always be borne in mind.

In the surgical treatment of duodenal ulcer excision of the ulcer is as desirable as in gastric ulcer, or, better still, the Balfour operation of artificial perforation by means of the cautery. The operation of choice in my estimation, however, is a pylorectomy followed by a gastro-enterostomy where there is wide-spread induration, and the ulcer is located in the first portion of the duodenum. As already indicated, pyloroplasty should be considered in certain cases. Excision is not always possible, especially when the ulcer is located low down in the second portion of the duodenum. In such cases invagination of the wall of the duodenum with the ulcer, followed by posterior gastro-enterostomy is indicated.

I now come to the consideration of *perforating ulcers*. In both gastric and duodenal ulcers perforation is always more or less of a menace, although at the Lankenau Hospital there seems to be a greater tendency to perforation in gastric than in duodenal ulcers. This, I know, is not in accord with other observations. Gastric perforations occurred in 20.4 per cent. and duodenal in 14.5 per cent. of the cases. This may, of course, be due to the fact that uncomplicated duodenal ulcer is today more easily recognized than gastric ulcer, and therefore is apt to come to treatment earlier.

The symptomatology of acute perforating gastric and duodenal ulcers does not differ much from that of perforation of any other abdominal viscus. Occasionally, a perforating ulcer may present prodromal symptoms in the form of sticking pain or an acute exacerbation of ulcer symptoms, but usually the onset is sudden, marked by violent, acute, overwhelming pain, without radiation, followed by vomiting, fall of temperature, increased pulse, peritoneal reaction, together with board-like rigidity of the abdominal wall followed by distention. The diagnosis of perforation is easily made, but it remains to determine what portion of the digestive tract is involved.

A definite history is rarely obtainable in these acute conditions, but the surgeon's first thought is usually gastric or duodenal ulcer. The question naturally arises why not think of the appendix first, since acute perforating appendicitis is comparatively so much more common than perforation of any other portion of the gastro-intestinal tract. The main reason is that peritonitis from acute appendicitis generally develops without the same gross signs of perforation as in ulcer, and without the sudden chemical irritation of the peritoneum. Anyone who has seen a number of cases of perforating gastric or duodenal ulcer soon becomes familiar with the typical board-like rigidity, which is rarely seen in acute appendicitis even with diffuse peritonitis, except in that small proportion of cases where the perforation is large and is located at the base of the appendix, at its junction with the cecum. The crouched, immobile position of the patient and, in the advanced stages, evidences of shock and air-hunger are pathognomonic. Of course, when the presence of a gastric ulcer has been suspected for some time the diagnosis in the presence of acute perforation will be easily made, but without this knowledge the differentiation between gastric and duodenal perforating ulcer is not easy. Since early operation is indicated in either or both, the distinction is not of much practical importance. As to other conditions, we have already mentioned the absence of board-like rigidity in acute appendicitis, and, in addition, it may be well to remember that in the latter, pain and rigidity are localized in the right iliac fossa and peritonitis develops more slowly. In a very few cases of perforation of an appendix lying in a high position the differential diagnosis may be impossible except by incision. This is also true of perforating ulcers that are seen some time after perforation, and when the right iliac fossa contains a large amount of the escaped gastric or duodenal contents.

The only other acute conditions with which perforating peptic ulcer may be confused are acute phlegmonous or gangrenous cholecystitis and acute pancreatitis. The latter, however, is comparatively rare, and the entire picture is so much more exaggerated than the perforating ulcer picture that the diagnosis is possible. Ruptured ectopic pregnancy may, but is not apt to be confused with perforating gastric ulcer. Acute intestinal obstruction has certain distinguishing features of its own, such as persistent vomiting, becoming stercoraceous in character, the absence of marked rigidity, and the relief of pain by pressure. Gall-stone colic and acute cholecystitis can usually be ruled out by the previous history and by the localizing symptoms.

That operation in these cases is imperative is obvious, the risks of operation being in direct proportion to the time elapsing after the onset of symptoms, to the size of the perforation, and to the amount of stomach contents at the onset of perforation. As to the type of operation, much depends on the state of the patient and upon the experience of the surgeon. The occasional operator may have to content himself with simple suture of the perforation, but the more experienced surgeon will, if he is wise, add a gastro-enterostomy and thus insure better results; while the specialist in abdominal surgery may even venture resection or pyloroplasty, the condition of the patient permitting. My experience with the radical operations—pylorectomy or resection together with gastro-enterostomy—in these cases has been most gratifying. A few years ago this radical procedure would have been thought as much out of the question for this type of case as gastro-enterostomy in the early days of abdominal surgery was considered too prolonged to be used as an emergency operation. I am aware that even today opinions vary with regard to the advantages of the gastro-intestinal anastomosis. Be this as it may, my records show that it does not add to the risk of the operation. I am able to point to a series of 54 consecutive operations for perforating gastric and duodenal ulcer with 100 per cent. recoveries, and in all but 2 of them a posterior gastro-enterostomy was done. In a series of 67 cases of perforating gastric and duodenal ulcer, 55 were treated by suture and the gastrojejunal anastomosis, with 3 deaths, or 5.5 per cent. mortality. The appendix was removed in many of these cases, and in 2 a cholecystectomy was also done. Of the 12 in which an anastomosis was not done, 2 died, making a mortality of 17 per cent. It is true that the latter group comprised the more desperate cases, but I find that other surgeons, advocating only closure of the perforation, report a much higher operative mortality, ranging, in fact, from 26 to 27 per cent. Thus I may say that the primary mortality is not increased by the more extensive operation, provided, of course, it is not done in the presence of severe shock or evident systemic toxemia.

A favorite argument against posterior gastro-enterostomy in the surgery of perforating ulcer is that opening the lesser peritoneal cavity presents the risk of infecting this cavity. I have neither heard of cases in which there is good reason to believe that it has occurred, nor have I myself experienced the circumstance. It must be remembered that the exudate in the early cases is sterile or nearly so, and the peritoneal reaction is a response to chemical irritation by the gastric and duodenal contents rather than the result of bacterial invasion. Another argument is that perforation of itself heals the ulcer. This is only partially true, for evidence is increasing that perforation of itself is not a guarantee of cure. Excision of a chronic ulcer by means of the Balfour cautery really produces an artificial perforation, but nevertheless gastro-enterostomy is generally performed in these cases.

One word as to technic: It is my practice after closing the perforation to plicate the duodenum, fortify the area by covering it over with gastrocolic and gastrohepatic omentum, and to complete the operation with a posterior gastro-enterostomy.

The end-results are available in 21 (40 per cent.) of the series of cases above referred to. Of these, 4 were treated with suture only, 2 with good results and 2 unimproved. Both of the latter returned some time later for a posterior gastro-enterostomy. Seventeen were treated by suture and gastro-intestinal anastomosis, with a complete cure in all.

The only valid argument against primary gastro-enterostomy in the treatment of perforating gastric or duodenal ulcer is the possibility of the development of marginal ulcer. The incidence of this must be low. I myself have not met with such a case, and I know of only one case reported in the literature, that of Petrèn, of Sweden.

Marginal Ulcer

Marginal ulcer, however, still figures as one of the sequelæ of posterior gastro-enterostomy in the treatment of chronic peptic ulcer. The exact etiology of these marginal or gastrojejunal ulcers is not as yet known. The trauma produced by the clamps used in the operation or by rough handling of the viscera may be a fundamental cause of this unpleasant sequel. Other points in the technic, such as the use of non-absorbable suture material, have led to the development of secondary ulceration in a certain percentage of cases. But even with the use of absorbable sutures there is no security against this event. Another probable cause of marginal ulcer, I believe, is faulty technic in making the anastomosis. When about to join the stomach to the jejunum it is of the utmost importance to grasp the anastomosis and hold the stomach and the duodenum well up, and to attach the jejunum to the stomach at a point about ¾ inch distal to the end of the opening. This is done to prevent the formation of a spur at the inner margin of the opening, so that the jejunal contents will pass uninterruptedly from the proximal to the distal limb of the jejunum. If this step of the operation is carelessly done and the organs are not held taut, there will result an unevenness or spur where the contents of the jejunum may collect, and by the action of the acid digestive juices produce a secondary ulceration.

But marginal ulcers may develop even in cases where every precaution has been taken to do a faultless operation. The impression is strong that in such cases an ulcer diathesis must account for the phenomenon. I have had one patient operated upon at three different times for recurrent marginal ulcer. This patient continues to have a high free and total acidity.

Occasionally latent infections, such as syphilis and tuberculosis, may also be included in the list of causative factors of recurrent ulcer after posterior gastro-enterostomy.

The ulcer symptoms usually reappear within a year of the primary operation, although there is often a history of relief or complete well-being for from three to five years after operation. The symptoms resemble those of the original disease, aggravated in certain cases by the presence of adhesions, and in others by indications of stenosis. Radiographic examination may show a change in the evacuation time of the stomach contents, but only in a few instances is the ulcer demonstrable. The history of ulcer treated by a gastro-intestinal anastomosis generally leads to a correct diagnosis.

Perforation of a marginal ulcer may take place and, as in certain types of duodenal ulcer, it may be the first indication of its presence. More commonly, however, there is a history of recurrence of ulcer symptoms.

In the treatment of marginal ulcer surgery, of course, is indicated, the type of operation depending upon the conditions found at operation.

In simple cases the old anastomosis can be resected and a new one made as near as possible to the old site. Sometimes more extensive surgery may be necessary. I have found it expedient in a number of instances to do a Roux-y anastomosis, and in others a subtotal gastrectomy combined with the Roux-y operation.

When the pylorus is patulous, excision of the anastomosis, closure of the opening in the stomach, end-to-end or side-to-side union of the bowel is the operation of choice. Where the pylorus is partially obstructed, pyloroplasty, in addition to the above, should be done.

Postoperative Treatment.—Except in emergency cases, in order to expose the patient to as little risk as possible from the operation, he will, of course, have been subjected to a thorough preoperative study, particularly as to the condition of the blood and of kidney function. It seems scarcely necessary to emphasize that the presence of acetone or diacetic acid in the urine contraindicates immediate operation. When, after suitable treatment. the surgeon is satisfied that operation can be safely undertaken, he will act promptly and according to his best judgment. Every surgeon should himself supervise the postoperative treatment. After the operation the first and most important considerations are: To control pain, to provide for the proper amount of sleep, and to guard against complications. If sleep cannot be obtained naturally, such medication as in the experience of the surgeon gives the best results should be administered. If in spite of the morphin that is being given for the pain the patient does not obtain the necessary amount of sleep, the best hypnotic, in my experience, is chloral, 30 grains, and bromid, 60 grains, given by bowel, in 2 ounces of milk, and repeated if necessary. The complication most to be feared is pneumonia; this can often be avoided, if I may put it this way, by giving atropin every hour in 1/50 grain doses and placing the patient on an open porch where he can have the benefit of plenty of fresh air. I have not found the administration of oxygen in these circumstances of much service; indeed, it is apt to be harmful, as it often is annoying to the patient and interferes with sleep. In the presence of rapid respiration and dullness upon percussion the affected side of the chest should be thoroughly cupped. The atropin should be continued in the above-mentioned dose until the chest is clear of mucus. Digitalis and whisky can be given by mouth if the stomach is tolerant; if not, a fat-free tincture of digitalis can be administered hypodermically, and whisky and creosotal in saline solution by bowel. This line of treatment has proven most satisfactory in my experience. Nausea, persistent belching, regurgitation, or vomiting call for lavage, repeated as often as is necessary. It is not my practice, as a rule, to allow water by mouth until the second day. Normal salt solution by enteroclysis, intermittent or continuous hypodermoclysis, may be used if needed. For acetone and diacetic acid in the urine I advise the use of glucose by bowel, which now seems to give better results than soda bicarbonate.

In using lavage it is a routine measure in the Lankenau Hospital to inspect the wound before and after the washing in order to see that no stitches are broken and that there is no prolapse of viscera. During the lavage the wound and the abdominal walls should be supported by the nurse. Disastrous consequences in the shape of partial visceral prolapse may result from straining during lavage, and oftentimes may fail to be recognized until too late to allow of a sterile replacement.

As to nourishment, I am guided by the retentive ability of the stomach, the absence of abdominal distention, and the presence of peristalsis. After convalescing from the operation the patient should be placed in the hands of a medical man. The time for medical treatment is after and not before operation, except in the absence of an experienced surgeon.

In view of the tremendous forward strides that have been taken during the past thirty or more years we have every reason to be proud of the achievements in gastric surgery during that time, and it is well nigh impossible to predict the future. But whatever it may have in store for those who come after us, the progress already made stands out as an enduring monument to those pioneer surgeons of the early days whose courage, skill, and foresight blazed the trail for us, their posterity.

PATHOLOGY

Dr. Deaver has mentioned the fact that gastric ulcers have been known for many years. Indeed, John Hunter described some of their characteristics. Cruveilhier's drawings in his "Traitè d'Anatomie Pathologique" can hardly be improved upon, and Rokitansky's descripton of their gross appearance is classic, and might with few if any changes appear in a modern text-book. He was familiar with the two chief dangers, namely, hemorrhage and perforation, and described how the risk of the latter is minimized by adhesions to the liver and the surrounding viscera, and why perforating ulcers situated on the anterior surface of the stomach are more serious in their immediate consequences. He also recognized the fact that carcinoma may arise in an ulcer. Virchow (about 1850), in his "Specielle Pathologie," describes the cellular pathology of ulcers, and theorizes as to their mode of formation. He leans strongly toward the theory that in some manner, not entirely clear, the pepsin and hydrochloric acid in the gastric juice are prime factors in their formation. Based on studies of the acidity of the vomitus, Bamberger, at about the same time, recommends the use of alkalies in the treatment of this disease, and goes a little more into detail concerning its etiology. Since that time descriptions of the pathologic anatomy, the incidence, and the more general features of the disease have appeared in enormous numbers, and with the advent of asepsis and modern operative technic the study of this condition was tremendously stimulated.

It might be profitable to spend a few moments in a general review of the pathologic anatomy of the subject. The position of the stomach in which the ulcers appear in the order of their frequency can be shown in the accompanying diagram which represents our experience in ulcers in both the stomach and the duodenum. Those in the latter organ are also included, since the duodenum at least in its first part is pathologically and physiologically similar in many respects to the stomach. For general purposes it is our custom to divide peptic ulcers into the acute, chronic, and those in which an acute exacerbation of a chronic condition was present at the time of removal. By far the greater number of peptic ulcers are single. In a total number of 143 specimens obtained in the laboratory during the last four years, all except 5 specimens presented one ulcer; 3 showed two ulcers, and in 2 specimens three ulcers were noted. The characteristic shape was rounded or oval, while in size they varied from ½ mm. to 3 or 4 cm. in diameter, the actual ulceration variously involving the mucosa, the submucosa, the muscularis, and the peritoneum.

A general description on which the classification of acute, chronic ulcer, and chronic ulcer with acute exacerbation is based, is as follows: The acute variety showed ulceration extending, in the majority of instances, through the mucosa, and part way through the submucosa. Peritoneal involvement in the deeper ones was shown by congestion and a flake or two of fibrin on that membrane. Microscopically, there was intense active congestion of the base and edges, and a small rim of necrotic tissue formed the immediate base. The neighboring parts showed more or less edema, cellular infiltration, and perhaps beginning repair. The chronic ulcers showed the classical crater-like appearance with

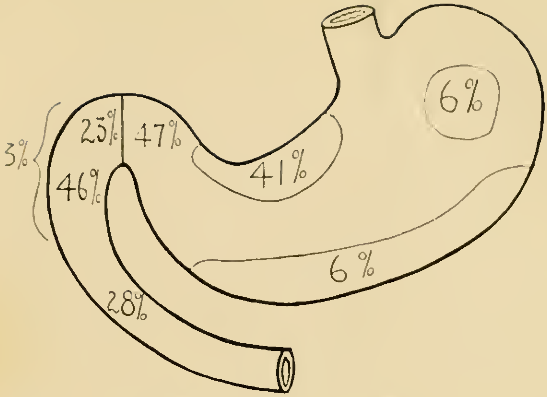

Fig. 4.—Location of ulcers.

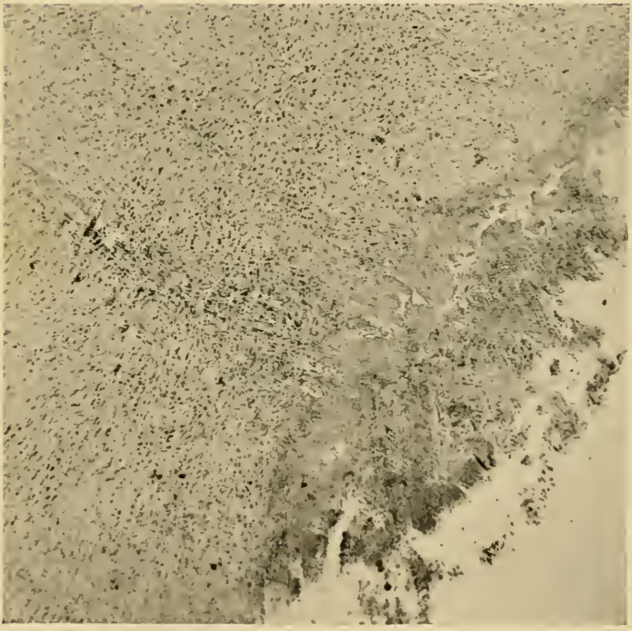

Fig. 4A.—The necrotic débris at the base of a chronic benign gastric ulcer, with the rich connective-tissue proliferation below it.

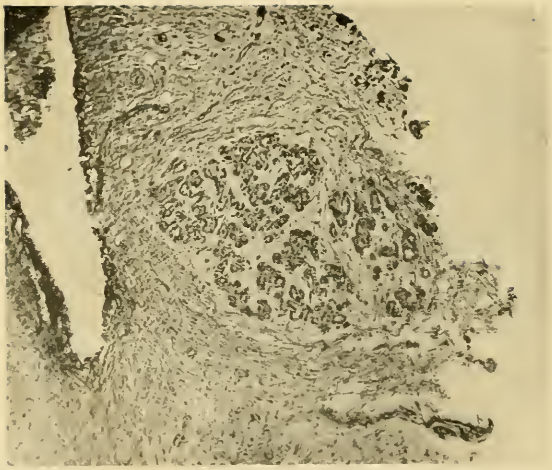

Fig. 5.—Section from the outer wall of a gastric ulcer on the posterior surface of the stomach, showing that the ulcer had infiltrated into the pancreas.

overhanging edges. Scar tissue had hardened the edges and base, and more or less congestion had produced hemorrhagic spots and thus influenced the color. Acute exacerbation showed grossly a much more decided congestion, swelling, and edema. The base was more ragged and more swollen, and the zone of reaction in the neighborhood was wider and more intense. With this as a background and with an emphasis on the fact that these ulcers in man tend to persist and progress for years, we may proceed to a discussion of a number of the phases of the subject which have especially appealed to us.

First, as to their etiology: From the very beginning of the study of peptic ulcers, the presence of acid gastric juice was looked upon with suspicion, and all manner of means have been tried to fasten the responsibility of ulcers on this substance. Intimately bound up with this question is the very obvious one which has puzzled investigators for years: Why is the stomach itself not digested? Perhaps it may be interesting to speak of some of the views regarding this point. Without mentioning names or quoting many authorities, a few of the hypotheses may be reviewed. John Hunter expressed the idea that all the tissues of the body showed an equal resistance to autodigestion, that while they were alive and uninjured, the ordinary digestive enzymes of the body could not dissolve them. This was accepted more or less generally until Claude Bernard performed his famous experiment in which he placed the leg of a living frog in the stomach through a fistula. The frog's leg was promptly digested. The conclusion followed that all tissues are not resistant to enzymes and that the stomach has some sort of a specific protective power. Later, however, better methods showed that although the frog was living, its leg was far from being uninjured, so that it was not the living protoplasm which was digested, but one previously devitalized by the poisonous action of the hydrochloric acid which had first killed the cells. A recent author makes the following dogmatic statement: "In the presence of living uninjured tissues digestive enzymes are inactive."

In the case of the stomach the problem is now concentrated to this: "Why is the gastric mucous membrane resistant to the injurious action of hydrochloric acid?" The answer to this question is not as yet available. It has been found that the natural gastric juice formed in the stomach itself with a high concentration of hydrochloric acid digests proteins much more quickly and more thoroughly than when artifically prepared with a similar concentration of hydrochloric acid, but—and here is the point—a natural juice acts less injuriously on living tissue than an artificial one. Natural gastric juice seems, therefore, less injurious than its content of hydrochloric acid would seem to warrant. This cannot be accepted as final, however, for a natural gastric juice which has no influence on the mucous membrane of the stomach will digest the skin when it flows over it from an open gastric fistula. We are, therefore, obliged to assume some sort of a specific property in the lining of the stomach which prevents injury by hydrochloric acid, and to accept this as a fact not yet clearly explained. Obviously more work is needed on this interesting and important problem. In such experiments as placing the leg of a frog, the ear of a rabbit, or in the later experiments, of implanting portions of the omentum, intestine, etc., into the stomach, the tissues, as a matter of fact, are injured in their nutrition before they are placed in the stomach. Injury, therefore, predisposes to the destructive action of hydrochloric acid, and in this respect the stomach mucosa is no exception to the rule. Now, can we assume that gastric ulcers may result from the corrosive action of hydrochloric acid and the digestive action of pepsin on a mucosa previously injured by some means or other? This question can probably be answered in the affirmative, at least in the case of certain ulcers. And the next question is, "By what means is the stomach mucous membrane so injured that a progressive ulcer results?" It must always be emphasized that the ulcers must be chronic and progressive. Nutritional disturbances following insufficient blood supply are unquestionably factors of the first importance. Experimentally, simple ligation of vessels, followed by excision or cauterization of a part of the gastric mucosa, almost universally failed to produce chronic ulcers. The wounds all healed promptly. Indeed, it has been found that four-fifths of the vessels of the stomach may be ligated without danger of gangrene. Since ligation proved unsuccessful, the next logical step was to interfere with the gastric circulation by capillary thrombosis. Various substances were injected into the gastric arteries, and it was found that relatively harmless bodies, such as India-ink and paraffin, produced no ulcers, but the addition of a toxic element to the injected material was sufficient to bring about the desired condition. Among these substances were formaldehyd, lead, etc. Finally, the general health was a very important factor. It was found that dogs which were kept under unfavorable circumstances, that is, given insufficient food, or kept in dark, damp cellars without opportunity for exercise, etc., were more liable to develop ulcers than those which were in good physical condition, and, furthermore, the presence of an infection of a general nature was a strong predisposing factor; thus dogs with distemper could easily be made to develop ulcers. Applying this knowledge to human beings, we can at the present time safely say that the following factors will be operative in producing the disease: poor bodily health, nutritional disturbances, and the destructive action of the gastric juice. But these nutritional disturbances must have a toxic element in them. The idea so often expressed by surgeons that an old or oft-repeated appendicitis with its toxemia may predispose to gastric ulcer has a foundation in this experimental work. Many authors, especially internists, believe that lesions in the vessels themselves, such as arteriosclerosis or even vascular spasm, may lead to sufficient nutritive disturbances.

What can we say of the discovery of certain bacteria in gastric ulcers as a causative factor? It seems to me that very little is proved by finding such ubiquitous organisms as staphylococi, streptococci, colon bacilli, etc., at the base of a gastric ulcer; it cannot be decided positively whether they were the cause of the condition or whether the infection was secondary. These ulcers are, of course, continuously surrounded by innumerable bacteria to which they offer suitable food-stuffs. On the other hand, Rosenow's work, much as it has been criticized, gives food for much thought, not to speak of the comfort it gives in considering many clinical problems.

Finally, the influence of the nervous system on the formation of gastric ulcers must be mentioned. Vagus resection, irritation or destruction of the celiac ganglion, or of the sympathetic nerves have all produced experimental ulcers, but rarely the progressive kind found in man. The view that reflex spasms of the blood-vessels are a cause has been recently revived and seems to be gaining ground. These reflex obstructions are said to be brought about by contraction of the muscularis mucosa which can be demonstrated, according to some, quite plainly by the *x*-ray. A number of interesting cases of ulcers developing in cases of lead-poisoning have also been reported, their important feature being that contractures of the ring muscle in the form of an hour-glass stomach were demonstrable by the *x*-ray long before an ulcer appeared. The interference with the blood-supply resulting from this spastic contraction was said to be the immediate cause of the ulcer in these cases. Recent studies in vagotony and sympatheticotony have brought out the fact that many such patients have constitutionally weak nervous systems, although, of course, the exact import of this is not clear. If, then, we sum up all these facts, theories, and opinions, we find that two points stand out quite clearly: first, ulcers develop on a degenerated soil. The exact cause of the degeneration in any given case must often remain open, although in some thrombosis, infection, anemia, etc., may be found; secondly, the hydrochloric acid and the pepsin certainly play some part in the progressive nature of the disease. It is, therefore, quite profitable to consider gastric and duodenal ulcers as "secondary diseases," and to leave no stone unturned in the critical examination of the patient and the eradication of all detected foci of infection, correction of habits of living, etc.

It has happened more than once that a patient who has had a gastric ulcer removed has developed another ulcer. Marginal ulcers following gastro-enterostomy, as Dr. Deaver has said, are by no means rare. Such causes as improper suture material, improper operative technic, etc., have been assigned for their development, but with the idea of "secondary disease" in mind, why cannot these be occasionally produced by the same cause which produced the first ulcer? Such an hypothesis is, of course, entirely logical in the case of the development of a new ulcer. Furthermore, this conception of "secondary disease" furnishes us with a fair reason for the chronicity of the ulcers, apart from the pathologic anatomic changes which will be mentioned shortly. Every ulcer must have had a beginning, and it may be said that it was an acute ulcer at one time. If the original cause was toxic, or nutritional disturbances, the acid gastric juice will dissolve the necrotic or devitalized area of mucosa. Attempts at repair will, of course, be made, but the same original cause not only makes the attempt at repair inadequate, but may also devitalize more tissue in the immediate neighborhood. The analogy to leg ulcers can be carried out to considerable detail. These develop notoriously in individuals with poor circulation in the lower extremity, and refuse stubbornly to heal until nutritional conditions are made better by rest in bed or other similar means. Once connective tissue has started to grow and has surrounded the ulcer, it will probably be an efficient factor in choking off nutriment and thus aid effectually in preventing healing. Assuming, for an instant, in the case of gastric ulcer, that the toxic circulatory condition had meanwhile been improved by, let us say, medical treatment, this hard, firm, calloused rim will certainly afford a most effective hindrance to healing even in the absence of all other disturbing factors. I need point only to the actual paucity of reports of autopsy specimens of actually healed scars of ulcers. According to certain statistics gastric ulcers are found in about 1 per cent. of autopsies. I do not know the percentage of healed gastric scars found at autopsy, but it must be quite low. It seems, therefore, arguing solely by inference, which sometimes is precarious, that if gastric ulcers do heal, they heal and leave no particular trace of their previous presence. On the other hand, that is a remarkable process which will remove the scar tissue in an ulcer of long standing, which extends over a large area of the stomach, without leaving some trace of its presence. That such processes do occur, however, seems probable. I can mention some very striking examples. We have had patients who came to operation for appendical abscess, and nothing more was done than to open the abscess, evacuate a large amount of well-encapsulated pus, and some necrotic bits of tissue, which may have been the appendix or parts of it. On account of the condition of the patient the appendix was neither searched for nor removed, but drainage was instituted and the patient allowed to recover. Some of these individuals have been reoperated later either for the repair of the hernia, which usually ensues, or for prophylactic removal of the appendix, or for a second attack of acute appendicitis. At the second operation it has frequently been found that the appendix which had been the starting-point and the site of a seemingly hopeless process, presented the appearance of having been little involved. Of course, there were anatomic signs that some sort of a process had been present, but these were remarkably few considering the past history of the organ.

Returning to gastric ulcers, the subject of gastro-enterostomy and its curative effect will be discussed a little later, but to anticipate, there have been large numbers of cases reported in the literature, and we have had a number ourselves in which a very large, hard, calloused ulcer was found at the first operation. On account of a suspicion of carcinoma the ulcer was not removed, but a posterior gastro-enterostomy was done in order to enable the patient to recover better health before undertaking a resection. At reoperation, four to six or more weeks later, in some cases all traces of the ulcer—it was, of course, not carcinomatous—had disappeared or it had been remarkably reduced in size. In spite of this, however, we cannot help believing that an ulcer will heal more readily before it has reached the calloused stage. Moreover, there is a remarkable form which we have encountered in 21 instances in our series of specimens. It consists of a circular, punched-out defect in the stomach wall, extending in some cases through to perforation, and in which there is a perfectly clean base, with absolutely no sign of inflammatory reaction around it. We have classified these as acute ulcers. Their appearance is exactly as though a portion of the stomach wall had been neatly and cleanly removed by a sharp instrument a very short time (no more than a few hours) previous to the reception of the specimen in the laboratory. The incidence of these ulcers is a most important question. Perhaps the majority of those which heal spontaneously or with medical treatment are of this type. If so, it would be a great stride forward in our knowledge of the disease as a whole, if we knew more concerning the incidence, the duration, the symptomatology, and the future of the patient with this particular type of ulcer.

The incidence of ulcer is a most interesting question to the clinician and the laboratory worker alike. Dr. Deaver has elaborated this phase in considerable detail. As stated, the condition is found at autopsy in a remarkably high percentage of cases. For instance, Osler says that among 10,841 postmortem examinations in the United States and Canada it was present in 1.32 per cent. of the cases; in Continental Europe it was found in 5 per cent., and in London in 4.2 per cent. of the cases. As a matter of interest, I have investigated my personal records of 600 autopsies performed at four different hospitals during the last seven years. Gastric and duodenal ulcers were found in 7 cases, making an incidence of over 1 per cent. Assuming then that the incidence in general is 1 per cent. (a low figure), it means that a comparatively enormous number of individuals have or have had gastric or duodenal ulcers. That much less than one in a hundred individuals complains of symptoms is a most remarkable fact. Let us for an instant consider one of the most characteristic complaints—pain. The question of its cause is still unsettled. There have been a number of theories proposed to explain it, but none has been entirely satisfactory. The most appealing one is concerning its relation to gastric contractions. Several observers have studied the movements of the stomach during the pain in ulcer and have found that the pain and contraction are often associated, whether the movements be of hunger or of digestion. This has not always been the case, however; pain has been present without definite contraction, and vice versa, but these studies nevertheless give a clearer light concerning its manifestation. Unfortunately, we are not very well informed of the pain nerves or the conditions giving rise to pain in the abdomen as a whole, and until further definite information is at hand this subject must remain in some doubt, but if approximately one individual in a hundred has a gastric or duodenal ulcer, whether or not he has pain will depend on the strength of the contractions of the stomach, the situation of the ulcer, that is, how close to nerve endings, probably the vagus, the psychology and general stamina of the patient, and so on. This subject, as can be seen, requires further extensive investigation. Needless to say, it must first be established that the study is really of ulcers. Dr. Deaver has emphasized the difficulties of diagnosis in upper abdominal affections, the *x*-ray notwithstanding, and I can emphatically endorse his views.

If we now pass to a consideration of the rationale of the common operative procedures for gastric ulcer, we must begin with a short consideration of some of the features of the normal physiology of the stomach. The organ may be divided roughly into three parts: the fundus, the antrum, and the pylorus. When food is taken, it enters the stomach, the walls of which, when the organ is empty, are in contact, and slowly wedges its way downward as the stomach actively opens. "Actively," because the dilatation of the stomach is not due merely to the weight of the food, but to an active participation by the muscles under the control of a nervous mechanism in the stomach itself. The food accumulates in layers in the fundus, and the masses last swallowed lodge in the center of the food bulk.

Liquefaction proceeds at the surfaces, and as the food is digested it is carried forward toward the pylorus. The fundus does not contract in actual waves, but rather presses steadily on the food, and thus forces it slowly into the antrum, where the movements are very powerful. In the light of newer research work our views on the opening and closing of the pylorus are undergoing a change. According to the chemical theory, putting it briefly, acid in front opens the pylorus, and acid behind closes it, but this idea, as elaborated by Cannon some years ago, does not seem entirely to conform to many of the facts. Recent work has shown that the opening and closing of the pylorus is quite intimately related to the rhythm of the antrum, so that at least these two factors are concerned. As digestion proceeds and the stomach empties, the greater curvature becomes raised so that ultimately the pylorus comes to be the most dependent part. This is not to say, however, that the emptying of the stomach depends at all on gravity. With regard to the secretory function, although this has probably been studied more than the motor function, it is not the better understood division. The gastric juice contains, of course, two important constituents—the hydrochloric acid and the pepsin. Secretion is controlled in two ways, first by the nervous system, and second by hormones. Psychic factors play a most important part. When palatable food is placed in the mouth, indeed, even at the sight or smell of food, the gastric glands secrete considerable quantities of the so-called appetite juice. This is quite important, for if foods are not relished, it is not secreted, and gastric digestion is proportionately interfered with. The second controlling factor is the so-called gastrin.

It has been found that the stomach with a small amount of partly digested food in it will continue to secrete even if all its nerves are severed. This fact, which led to much speculation and theorizing, was finally related to the absorption and action of a substance formed in the stomach in the presence of food partly digested by the appetite juice. Although this so-called gastrin has been the object of many investigations, the conditions concerning it are not very well understood, nor need they be discussed here.

The operative procedures may now be briefly considered. It is not difficult to understand that excision will remove a gastric or duodenal ulcer, nor is it difficult to understand that a gastro-enterostomy will occasionally be necessary to take care of the obstruction produced by the removal of tissue and subsequent suture, but that a gastro-enterostomy alone should heal an ulcer is more difficult of comprehension; in fact, the rationale is not at all clear. It was thought at first that it led to a more rapid evacuation of the stomach contents, but this has been shown to be erroneous. The rate at which the normal stomach empties depends for the most part on the type of food ingested. According to Cannon, fats are discharged slowly, proteins a little more rapidly, and carbohydrates the quickest of all. The discharge of food usually begins after the first half hour, although a small amount may leave before that time. Suitable studies have now shown that after gastro-enterostomy the rate of emptying is very little influenced, that is, the food does not leave the stomach very quickly by simply dropping through the gastro-enterostomy opening. When the stomach is empty the gastro-enterostomy opening is probably closed. The filling of the operated stomach proceeds as in the normal organ; after a good-sized meal has been eaten, that is, at about the same time as in the normal stomach, some of the digested food passes into the bowel. The contents, however, do not leave in a continuous stream, but in jets, exactly as through the pylorus. This occurs even when the opening is located in the fundus and not toward the pyloric end of the organ. When the gastro-enterostomy opening is in the antrum the contents flow out to the rhythm of the antrum movements, when it is in the fundus the food leaves the stomach in irregular squirts. Attention is again brought to the normal movements in which it is shown that the weight of the food plays no part in the emptying of the stomach. Since there is no ring-shaped muscle at the opening it must be assumed that its periodic opening and closing is due to the peristalsis of the stomach and the attached loop of bowel. We have always believed, and it is always taught, that when the pylorus is patulous very little food will pass through a gastro-enterostomy opening, most of it leaving by way of the pylorus. During the last two years we have had the opportunity of examining fluoroscopically the conditions after operation on all of our ulcer patients who returned to the follow-up clinic. Through the courtesy of Dr. Miller, of the *x*-ray department, I am enabled to add this interesting observation. It is true that in most of the patients who have had a gastro-enterostomy performed for gastric ulcer without disturbance of the pylorus most of the barium passed through the pylorus and none through the gastro-enterostomy opening. Indeed, in more than half none at all passed through the gastro-enterostomy opening, so that we were inclined to believe it might have closed, but as patients returned for second, third, fourth, and fifth observations at varying times after the operation, it was found that some whose stomachs had emptied completely through the pylorus at one examination, showed, on later re-examination, that all or nearly all of the food passed through the gastro-enterostomy opening. The reverse was also found in several instances, namely, that at one examination the barium passed through the gastro-enterostomy opening and none through the pylorus, whereas later it passed through the pylorus and none through the gastro-enterostomy opening. It is at once apparent that the conditions require much more investigation. In the future, however, we cannot assume that the gastro-enterostomy opening has closed, or that it never functions simply because at one examination it is found that no barium passes through.

While, therefore, the change in the rate of emptying is not markedly affected, the chemistry of the stomach is said to be quite considerably changed. There is, first of all, a regurgitation of alkaline intestinal juice into the stomach which reduces the acidity. The activity of pepsin is immediately diminished, or, if the reaction becomes alkaline, it is stopped. But this does not interfere materially with the digestion of food, for the protein, fat, and carbohydrate-splitting enzymes of the pancreatic juice, while they act best in an alkaline medium, are resistant to acid, and are thus able to carry on their functions in such a medium. Thus after gastro-enterostomy, digestive processes take place in the stomach which otherwise occur only in the bowel. That digestion as a whole is efficient can be demonstrated by the failure to find changes in the total metabolism after gastro-enterostomy. Another factor which might incidently be mentioned also tends to stabilize metabolism. It is said that the passage of food in the small intestine is slowed after gastro-enterostomy, and this possibly helps to assure normal comminution and absorption.

Another question which arises in connection with gastro-enterostomy with an occluded pylorus is, since food no longer passes over the papilla of Vater, are the pancreas and liver stimulated to secrete as before? The principal stimulus to pancreatic secretion is the so-called secretin which is formed from the mucous membrane of the duodenum with the help of the hydrochloric acid. Since secretin may also be formed in the jejunum, indeed, very recent work seems to show that it can be formed in other places, for example, the stomach, the flow of pancreatic juice after gastro-enterostomy is probably not disturbed. Furthermore, at least in animals, there is considerable backflow of fluid over the papilla of Vater. This probably takes place in human beings also. It is, however, observed principally in connection with the so-called vicious circle. This latter condition, as is now well known, depends on faulty mechanics at the time of operation and need not be discussed here.

In view of the foregoing observations two factors must, therefore, be considered in the explanation of the undoubted healing action of gastro-enterostomy. We have stated that excision, etc., is not difficult to understand. We have seen that the changes in the motor function in comparison with the normal stomach are of minor importance, but we must remember that in a large number of cases coming to operation for ulcer the pylorus is partially or totally obstructed not only on account of the presence of an ulcer and its scar tissue, but because of a pylorospasm produced reflexly by an ulcer in another part of the stomach or duodenum. In both cases the gastro-enterostomy opening acts purely mechanically and undertakes the emptying of the stomach until the ulcer heals, and thus the obstruction to the pyloric opening is removed. In ulcers at a distance from the pylorus it has repeatedly been observed that pylorospasm disappears after operation. The better evacuation of the stomach alone may not completely explain the repair of ulcers after gastro-enterostomy, since, of course, at least one injurious factor — and that a very important one, namely, the action of digestive juice—acts on the ulcers after operation; but we have seen that the second factor—change in the chemistry of the stomach—is quite important. The hydrochloric acid is neutralized by the backflow of alkaline intestinal contents, and its necrosing effect on the cells of the stomach wall is proportionately diminished. Since the intestinal fluid hardly reaches all parts of the stomach, but acts principally in the neighborhood of the pylorus, not only because most gastro-enterostomies are made in the region of the antrum, but because any backflow would be forced toward that end of the stomach by the movements of the organ, it is easily understood why after gastro-enterostomy ulcers on the cardia or on the lesser curvature heal with more difficulty than those at the pylorus. Probably on account of this there are more surgeons who prefer resection, excision, etc., of ulcers distant from the pylorus than those who prefer this procedure for ulcers at the pylorus. Dr. Deaver has elaborated his views on the subject in considerable detail.

There remains one other phase which merits considerable discussion, and that is the relation of carcinoma to ulcer. As usual in medical literature on subjects in which viewpoints and opinions play a large part, statistics vary considerably. There are those who believe that all carcinomata are such from the very beginning; there are others who believe that 70 per cent. or more of carcinomata arise in a pre-existing ulcer. But there is more uniformity of opinion as to the relation between chronic irritation and the arising of carcinoma. In fact, one might say that it is now accepted without much question that chronic irritation may lead to carcinoma. In all standard text-books, viewing the subject of carcinoma as a whole, without reference to any specific part of the body, among predisposing causes chronic irritation is practically always mentioned. Virchow considered this a most important cause, and there has been no great advance in our knowledge since his time. Even the nature of the irritant is still as vaguely spoken of as before. It may be mechanical, chemical, thermal, toxic, bacterial, and so on through a very long list, but how it acts or through what means or under what circumstances cannot be told. It would seem that if traumatism (irritation) were an essential thing, it should be possible to reproduce tumors with considerable ease in animals, but it can be said unequivocally that with the exception of a few unusual types of growths, not tumors in the strict sense of the word, no one has as yet succeeded in producing a tumor at will. Furthermore, everyone is continually being "traumatized" or "irritated" somewhere or another, and yet not every one develops carcinoma. In a gastric ulcer reparative processes are going on nearly all the time in addition to destructive processes. Repairs consist in the elaboration of new tissue to bridge defects, etc. When this reparative process becomes excessive and the tissue keeps on growing, we have what is known as a tumor. When I say what is known as a tumor we must recognize that what is a tumor to some is not a tumor to others. In fact, when we begin to define the term "tumor" we meet with almost insuperable difficulties because the fundamental thing of what constitutes a tumor is not known. In gastric ulcers it is not only the connective tissue which proliferates, but the reparative process is shared by epithelium, since we know that gastric ulcers heal and the defect in the mucosa is covered over by new epithelium. If this epithelial growth is slightly excessive at one place and remains there, it is not a tumor, but if it invades beyond the zone of inflammation and repair, it becomes a carcinoma. Suppose, in a number of histologic sections, we have a picture showing a very large amount of connective tissue close to the edge of an ulcer in which are embedded epithelial cells which appear histologically in the "active" state. Suppose we search by means of many sections through the entire circumference of the ulcer, in the neighboring lymph-nodes, and in the remainder of the wall of the stomach, and find apparently abnormal epithelium only toward the surface of the ulcer, but still embedded in the connective tissue of repair. Is it there because it was "snared off" by the growing connective tissue, or did it grow into the connective tissue in an effort at repair? Is it there actively or passively? Is it a carcinoma or is it regeneration? Obviously, if those epithelial cells were multiplying to cover over the defect in the mucosa, they should appear to the histologist's eye as active. If they had decided to keep on growing and to invade and infiltrate the deeper tissues, but had been removed by the surgeon before they had the opportunity, *i. e.*, the time to invade, to the histologist's eye they would still appear active, but in this case they would, in reality, be carcinoma cells. But how is the histologist to know what they would have done if the surgeon had not removed them? Suppose they were carcinoma cells, and the surgeon, fortunately for the patient, had applied his knife when the process was just beginning and had removed all of the abnormal cells, how are we to tell that this was a carcinoma if the patient remained well and presented neither a return of the tumor nor metastasis? On the other hand, if they were only intending to grow and cover the defect, how were we to know that? Let us look at the question from another viewpoint. It is remarkable, when one comes to consider it, that the offspring of human beings should always be human beings; that the offspring of dogs, dogs; pigs, pigs, etc. It is also remarkable that while monsters are occasionally produced, the overwhelming majority of individuals, be they human or otherwise, are born well formed with, let us say, the skin on the outside and not underneath. If we cut through skin and subcutaneous tissue we will find a sharp line of demarcation between the two. Is the epithelium prevented from growing downward by lack of energy, or because of an insuperable resistance opposed by the underlying tissue, or both? If the epithelium takes on an increased energy of growth, it may grow upward on the surface and produce what we know as a papilloma, if, because of increased energy of growth, decreased resistance of subcutaneous tissue, or both, the epithelium grows downward, we speak of it as a carcinoma. There is involved in this question the one of growth and reproduction, and when all is said, is this not the most fundamental question in biology? No wonder we know so little about it.

But perhaps I am making the subject too difficult. Perhaps I am drawing too fine lines. It is true enough that the average carcinoma can be readily diagnosed, but it is not true that we can be sure that it arose from a previous ulcer. Neither does the gross appearance give any definite assurance, for carcinomata in the stomach oftentimes ulcerate. They are frequently of a scirrhous type, that is, contain considerable connective tissue, and in many instances it is practically impossible to decide from the specimen alone. For help, we may fall back on the history of the patient. The following is the type: The patient has had "stomach trouble" for years, coming in attacks lasting three to four weeks, at intervals of six to eight months. Appropriate treatment, or no treatment, has resulted in relief, until at one time instead of relief being obtained there was progressive increase in the symptoms. At operation a carcinoma is found and removed. Pathologically it is of the ulcerative type grossly, and the small-celled scirrhous type histologically. This gives us much better presumptive evidence that the ulcer had been there for years and that carcinomatous transformation had occurred. Our records contain one very remarkable case in this connection in which the patient had had this type of stomach trouble for thirty years. For the past five years he had had trouble of a decidedly

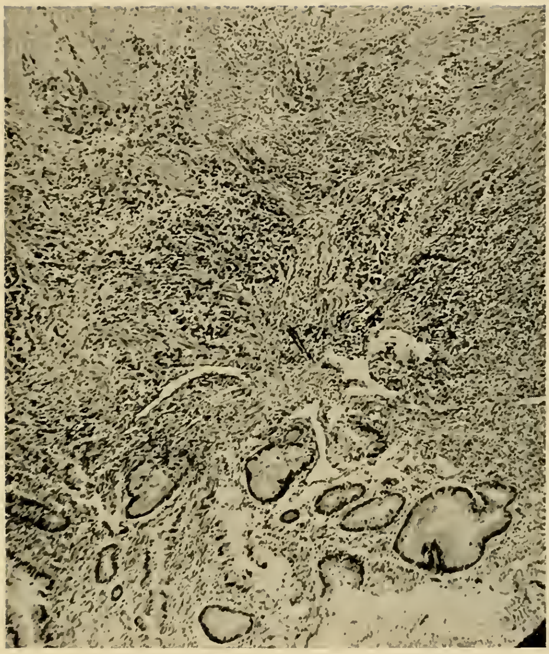

Fig. 6.—Carcinoma cells streaming into the outer layers of the stomach from a large ulcer in the mucosa. The section shows a few remnants of the old mucosa and a small portion of the submucosa. As indicated in the text, we are not certain whether this was primarily an ulcer and secondarily a carcinoma, or a carcinoma from the very beginning.

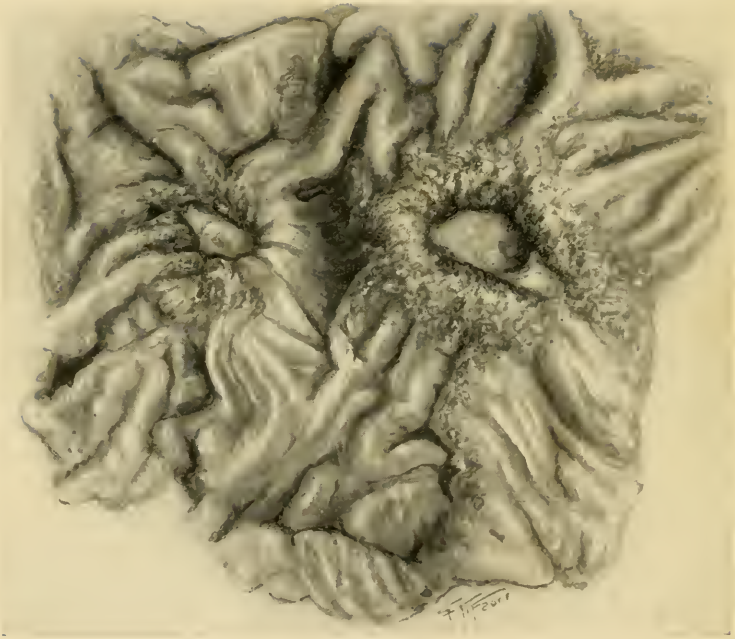

Fig. 7.—Portion of the stomach removed at operation showing two ulcerated areas, the large one being carcinomatous.

more severe type, but always obtained relief after rest in bed, carefully chosen diet, and alkalies. The last attack failing to yield to these measures, operation was decided upon. Two ulcers were found toward the pyloric end of the stomach; one approximately 2 cm. in diameter, very hard, and scarred, with carcinoma in it; the other about 1 cm. in diameter, obviously chronic, but not as hard nor as indurated nor as extensive as the other, with no carcinoma in it. Did the patient have one ulcer for twenty-five years; two ulcers from that time onward until the recent attack, when one of them became carcinomatous, or was the one ulcer present for the entire thirty years and the carcinomatous one a recent affair and carcinomatous from the beginning? We shall never know; but a lymph-node received and some gastrohepatic omentum showed carcinoma, therefore we assume that the patient's chance for complete recovery is slight. To sum up, therefore, we are not certain from either the gross or the histologic appearance of a carcinoma whether it arose in a previous ulcer or not. The history, to our mind, is of prime importance, but considering the difficulties of diagnosis and the other difficulties in reference to healing, etc., which we have outlined in some detail, we must accept this evidence as contributory and not positive. With these ideas in mind, a serious attempt to analyze our cases of carcinoma of the stomach resulted in the conclusion that there had been an ulcer present in some 35 per cent. of the cases. Whether these actual figures are correct or not is relatively unimportant, but it is safer from the patient's point of view to err on the positive side and to say that carcinoma may arise in a gastric ulcer; how often, we do not know, but probably sufficiently often to make it advisable to remove the ulcer wherever practical.

Finally, in the latter part of this discussion I have brought up the idea of correlating the particular patient with his specimen, and I cannot miss the opportunity of emphasizing the importance of this thought. If pathologists working in this line are not given the opportunity of knowing the patient not only before but also after the operation for an extended period, they will be working under difficulties which are to say the least, crippling. This is the age of early diagnosis and early removal of tumors. The pathologist receives specimens earlier than he did, let us say, twenty-five or thirty years ago. It is not difficult for a competent pathologist to diagnose outspoken carcinoma or sarcoma or other such tumors, but it is the borderline case that presents difficulties. Pathologic anatomy is, of course, an art as well as a science. I am inclined to believe it is more of an art than a science, for while it is possible to draw up general rules to be used in teaching, it takes but a few months' experience to discover that very few specimens, especially histologic ones, conform to rules. Anyone who has taught pathology will know of the difficulties and the work necessary to collect specimens "typical" of any lesion so that students may be taught the fundamentals. One specimen will conform more or less to the rules, and ten will not. Pathologic anatomy is, therefore, more of an art than a science, and when a pathologic anatomist casts his eye on a section, in forming his final opinion, he brings to bear, a series of scientific reasons plus an association of many memory pictures of previous specimens, where they came from, how removed, and what happened to the individual after removal. Unfortunately, this latter and most important phase has not been developed as it should be, not because of any fault of the pathologist, but more because the surgeon has failed to supply him with data. The surgeon has the mistaken idea that a pathologist's opinion is based on a glance at the picture as observed through the microscope. This may be so in certain instances, perhaps in most instances, but in those cases there is usually no doubt of the diagnosis from the clinical and gross aspects. Since the surgeon has advocated early and prophylactic removal of tissues, borderline cases are occurring more often, and it is especially in these that it is necessary, in order for the pathologist to give an intelligent opinion and to guide the future therapy, that he have complete memory pictures of previous specimens of a similar nature, what happened to the patients who had them, and what therapeutic measures were directed toward their future. The advent and much more general use of x-rays and radium in both the pre- and postoperative treatment of neoplasms has added another factor which the pathologist must take into consideration. x-Rays and radium produce queer pictures when applied to tumors. All sorts of bizarre conditions are found. In judging a tumor, especially in regard to its behavior under treatment by the x-rays or radium, such future history is essential. We cannot help regarding it as a very legitimate field for pathologists, that is, not the technical details of the application of x-rays or radium, but the acquisition of the knowledge of what will or will not happen to a tumor either *in situ* or after it is removed, and the probability of its return. These last remarks are simply a plea for closer co-operation between clinicians and pathologists, and I hope I have shown at least a few of the important reasons for such teamwork.

JAUNDICE

It is only within the past three decades that the true nature of jaundice has been recognized and its importance appreciated. Before that time the prevalent belief was that it was due to some mechanical obstruction to the flow of bile. Indeed, the revelations of abdominal surgery seemed to support this theory, for calculi and cancerous and inflammatory diseases were continually demonstrated as obstructing factors. These however, failed to account for the many instances in which no direct obstruction to excretion was present, as, for example, in acute yellow atrophy of the liver, Hanot's disease, the hemolytic anemias, phosphorus-poisoning, and various infectious diseases and toxemias. It was then realized that jaundice is not a clinical entity, but a symptom which may accompany a variety of pathologic conditions, and therefore could be relieved only by the successful diagnosis and treatment of the disease itself. Thus a distinction arose between obstructive, catarrhal, and hemolytic jaundice.

The term "jaundice" indicates the staining of the body tissues with bile-pigments as the result of their presence in the circulation. In normal conditions these pigments are manufactured principally in the liver, and are present in demonstrable amounts only in the bile. The secretory pressure under which the bile flows outward is quite low, equalling only about 200 mm. of water, so that it is easily overcome by the slightest disturbance in the liver or in any of the bile channels. In the presence of obstruction, either intrahepatic or extrahepatic, the bile is dammed up, the ducts dilate, the capillaries distend, and finally rupture into the adjoining lymph spaces. Bile is then absorbed by the lymphatics and is carried to the circulation by way of the thoracic duct. The manner of absorption in cases of non-obstructive jaundice is not so clear, but this also, according to Eppinger, may be similarly explained as the result of obstruction, stasis, and dilatation in the minute radicles of the biliary channels. Eppinger's ideas were based on morphologic grounds, but others believe that the icterus is due rather to a disturbance in the function of the hepatic cells themselves which results in the excretion of biliary constituents directly into the blood-stream.

The most common type of jaundice is that due to partial or complete obstruction of the common bile-duct. Complete obstruction is usually the result either of tumor formation at the papilla of Vater, at the orifice of the cystic duct, or in the head of the pancreas, since the ends of the common duct and the pancreatic duct often run parallel, or it occurs as the result of stone formation. While it is possible for small stones to pass through the papilla of Vater, the larger ones are apt to lodge at this point, favored by inflammatory processes initiated by their presence. As a subthought, complete obstruction may also result from outside compression, particularly from neoplasms, either primary in the bile-ducts or metastatic infiltration; for it is a well-known fact that the numerous lymphatics along the course of the hepatic vessels are favorite sites of carcinomatous infiltration. Compression from syphilitic processes, as well as from the scars of duodenal and gastric ulcers, have also been known to cause complete

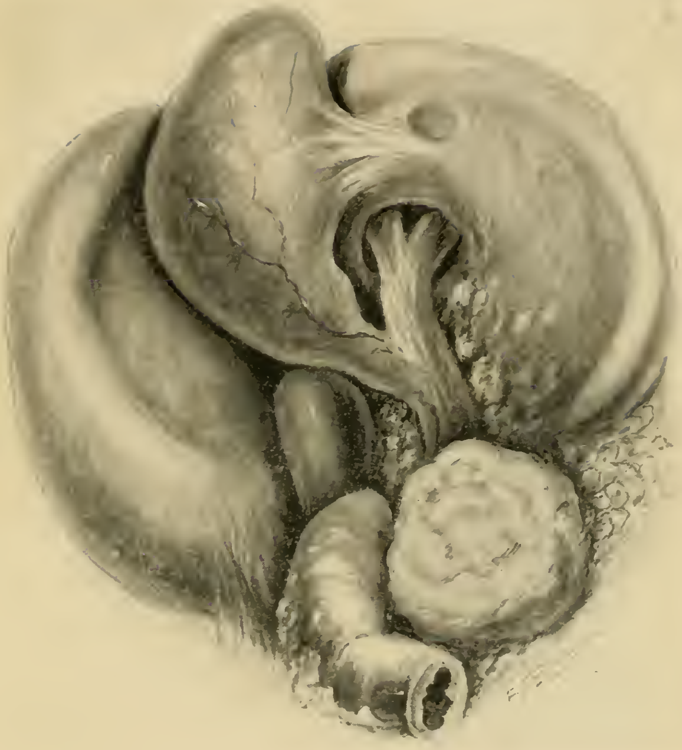

Fig. 8.—Autopsy specimen removed from a patient who had complained of illness only four weeks, during which time an increasing jaundice developed, but no pain until a few days before his death. The head of the pancreas is shown enlarged, with a carcinoma constricting the common duct. There was only one small metastatic nodule in the liver to the left of the gall-bladder. The biliary passages were enormously distended, and contained fully a liter of bile. It is interesting that the body and tail of the pancreas which were not carcinomatous, showed a very marked sclerosis and replacement of the parenchyma, just as occurs in experimental ligation of the pancreatic, ducts. The islands of Langerhans were, however, well preserved microscopically, which also corresponds to the experimental finding that these structures remain preserved for a considerable length of time. The man showed no glycosuria or hyperglycemia.

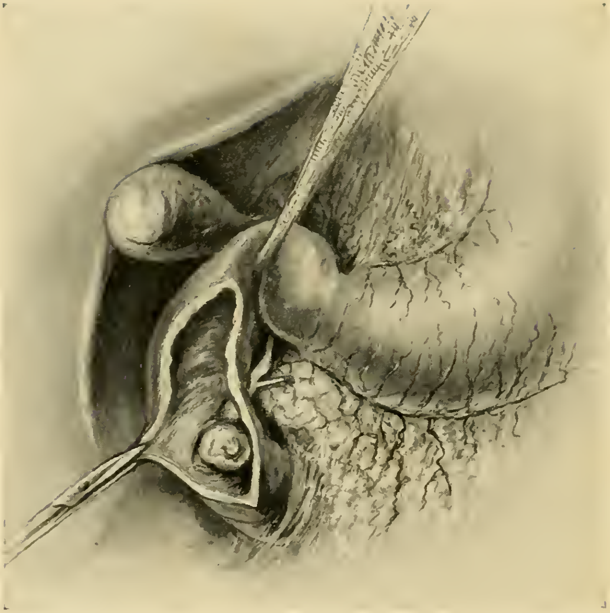

Fig. 9.—Protrusion into the duodenum of the papilla of Vater occasioned by the presence of a stone in the papilla.

obstruction. Of course, partial obstruction may be the result of any of these factors, or it may be due to cholangitis, as well as to the presence of a stone or stones in the hepatic ducts. Finally, the obstruction may be intermittent, especially in cases of cholelithiasis or in the presence of a common duct stone with a ball-valve action.

Although the presence of bile-pigments in the circulation may be comparatively well tolerated in some cases, the resulting yellowish hue of the skin and the mucous membranes should call attention to the presence of some fundamental causative disorder, although it is true that a subicteroid hue may be due to a mild transitory cholangitis and a true jaundice may never develop. Generally, the onset of the obstructive type is gradual, the yellowish tinge of the skin and the sclera is at first very slight, but progressively increases in intensity, according to the degree of obstruction. A sudden deep jaundice is rare except when a large calculus suddenly blocks the common duct.

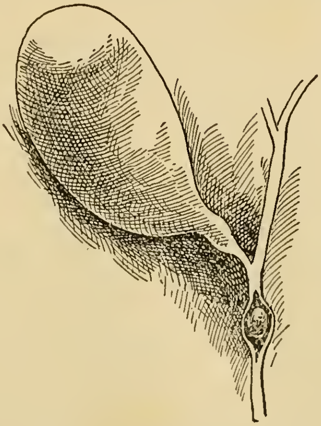

Fig. 10.—Stone in the common duct, showing the mechanism of ball valve action.

The degree of jaundice is not an indication of the cause, although it may usually be taken for granted that if progressive and persistent, it is, in all likelihood, due to a malignant process.

The most constant feature of icterus is itching of the skin, more or less severe, but there is no relation between its severity and the degree of jaundice. Very severe and even fatal cases may progress without pronounced itching. Its cause is not definitely known, although it is generally attributed to the presence of bile salts in the tissues.

Discoloration of the urine is another symptom, due to the excretion through the kidneys of the bile-pigments present in the blood. This usually precedes the pigmentation of the skin by a few days and is often the first clinical sign. The urine may at times be reduced in amount and its specific gravity increased. Acholic or clay-colored stools are pathognomonic of obstructive jaundice, being due to the absence of bile-pigments; variations in the color of the feces occur according to the intermittent nature of the flow of bile. When obstruction is complete, as from a tumor in the head of the pancreas, the stools are constantly devoid of bile and are of a grayish color. They show an increase in undigested fat, which may be as much as 80 per cent. above the normal. Frequently the obstruction which prevents the bile from entering the duodenum compresses the pancreatic duct also, thus blocking the flow of the fat-digesting pancreatic secretion. The fat present in the stools is, for the most part, in the form of fatty acids and soaps, and the odor, according to Schmidt, is due to the fatty acids becoming rancid instead of putrefying. When obstruction is not complete and bile can be chemically demonstrated in the feces, they may be clay colored because of the large amount of fat which obscures any other color present. When there is also obstruction of the pancreatic duct, they sometimes contain undigested protein, which gives them a decomposing odor, and may produce catarrhal conditions; 50 per cent. or more of the ingested protein may thus escape absorption instead of the small amount normally excreted in the feces. This may be the reason why some patients with complete obstruction of bile associated with pancreatic involvement are apt to complain of diarrhea, while in simple obstruction of the biliary passages alone constipation is more generally the rule.

In view of the changes in the blood in jaundice the question of priority of cause and effect must be considered. Since Whipple and Hooper have shown so clearly that bile-pigments may be formed rapidly and in considerable quantity in other parts of the body, it is possible that a true hematogenous jaundice may occur, but liver damage must surely play a more or less important rôle in the jaundice of the hemolytic anemias. After an attack of catarrhal jaundice the blood-count usually shows a decrease in hemoglobin and in the number of erythrocytes, and there is usually an increase in the coagulation time, generally from three to four and sometimes as much as fifteen minutes above the normal. Slowing of the pulse should be mentioned, in passing, as a fairly constant accompaniment, and the work of Stewart and King seems to indicate that it is due to the action of the bile-pigments in increasing the tone of the vagus nerve.

It must be borne in mind, however, that disease of the bile-passages is not necessarily associated with jaundice, nor is every case of jaundice to be interpreted as a disease of the bile-passages, or as the result of gross mechanical obstruction to the flow of bile. There are two large groups of disorders in which the ducts are patulous, as manifested by the presence of bile or its derivatives in the stools, but in which the parenchyma of the liver is involved. The first of these groups includes catarrhal jaundice, the infectious disorder known as Weil's disease, syphilis of the liver, acute yellow atrophy, jaundice due to poisons, such as arsphenamin, and finally, cirrhosis of the liver. The second group comprises the types of hemolytic jaundice represented mainly by the pernicious anemias and familial icteroanemia, and, less prominently, by malaria. Catarrhal jaundice has been placed in the first group, but not without some hesitation. Certainly the theory accepted for so many years that it is obstructive, due to an ascending inflammation of the ducts, seems logical, especially since the first symptoms are often some gastrointestinal disturbance and the passage of clay-colored stools. On the other hand, slight fever, tenderness over the liver, together with dark urine and normal stools may be the initial symptoms. In such a case it is possible that not so much an obstruction as a functional disturbance of the hepatic cells themselves is the cause of the trouble. There is, however, considerable controversy on this point, and rightly so. The lesions in the milder types of catarrhal jaundice are probably slight and of short duration, as can be determined by special tests for the amount of bile retention, by examination of the urine, the feces, the blood, and the duodenal contents.

In this connection it may not be out of place to mention dissociated jaundice, a favorite subject of discussion by French authors. As indicated by the name, in this type, a dissociation of the bile-salts and the bile-pigments takes place. In other words, the bile-salts formed within the liver are capable of being sent on independently into the circulation, but whether the bile-pigments may also enter the blood separately is not definitely known. The French have used the term "complete jaundice" for those cases in which both the salts and the pigments are present in the blood, and "hepatic dissociation jaundice" when only one of these is found, while the term "hemolytic jaundice" indicates the presence of the bilirubin alone when this substance is manufactured extrahepatically. To the surgeon it is this last group that is of particular interest because it includes hemolytic jaundice, pernicious anemia, Banti's disease, etc., all of them complexes, which are very imperfectly understood.

In hemolytic jaundice the discoloration of the skin is due primarily to the increased fragility of the red cells with subsequent disintegration and formation of bile-pigments. This phenomenon was formerly supposed to take place independently of the bile-forming function of the liver, but the investigations of a host of authors have established a close relationship between hemolytic jaundice and the behavior of the liver cells. In fact Rost, Eppinger, and others have found an analogy which they explain in this manner: As a result of their diminished resistance (probably the fundamental cause of hemolytic icterus), the red blood-cells are destroyed by the spleen in large numbers and the liver is inundated with pigment which causes an increase in the viscosity of the bile, producing inspissated plugs in the small bile-ducts. Since the pressure is not sufficient to send the bile onward, it accumulates and is forced into the lymphatic system, and thence into the blood-stream, with icterus as the result. In other words, there is the same thrombosis of the bile capillaries as in the obstructive type.

Hemolytic jaundice occurs both as a congenital familial disease and as an acquired disease of adult life. The first type is the more common one. Chauf-ford, in 1914, drew attention to syphilis and tuberculosis as fundamental factors, but this idea cannot be accepted unequivocally, for, although these chronic infections were present in a number of Chaufford's cases, there are numerous instances on record in which they can be positively ruled out as causative or even concomitant factors. He aptly describes his patients as being more icteric than sick. On the other hand, the relationship between hemolytic jaundice and disease of the liver and gall-bladder is conclusively proved by the observations in the Mayo Clinic, which show that 60 per cent. of their cases were complicated with cholelithiasis. According to their suggestion, the predisposition to the formation of gall-stones is due to the viscosity of the bile caused by the flooding of the liver with blood-pigment set free by the destruction of numerous red blood-cells in the spleen. This also may lead to cholangitis and finally to cirrhosis of the liver. The fundamental characteristics of the disorder are splenomegaly, acholuric jaundice, and anemia of varying intensity. This increased red blood-cell destruction is manifested by urobilinuria and by marked changes in the blood. The erythrocytes show a diminished resistance to hypotonic salt solutions, and in the acquired form the phenomenon of auto-

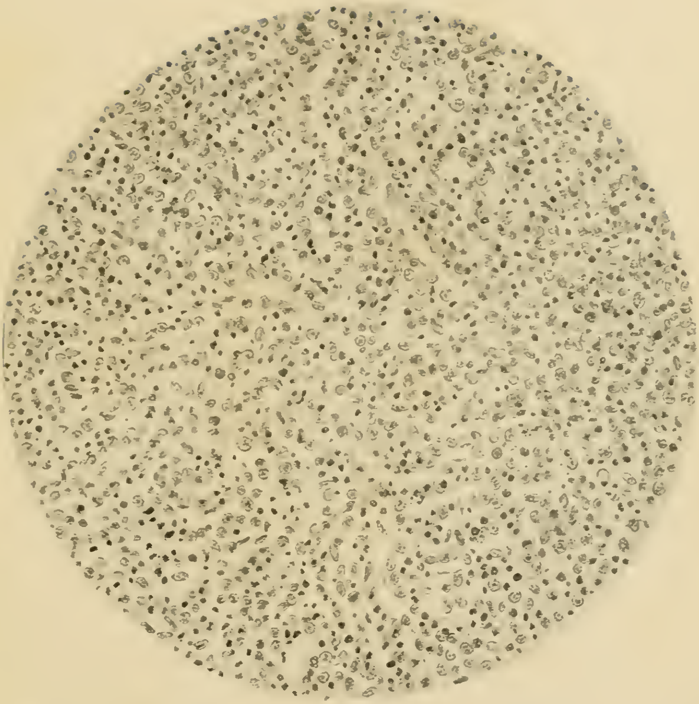

Fig. 11.—Drawing of a section of a spleen removed from a case of hemolytic jaundice, showing the enormous amounts of intra- and extracellular blood-pigment granules.

agglutination can be noted. Krumbhaar attributes the pathologic changes in the blood-picture to abnormal blood destruction, while in Banti's disease and in Gaucher's disease they are due chiefly to impaired blood formation. It is the anemia, however, for which relief is generally sought. The splenic enlargement usually passes unnoticed and the jaundice is often merely a sallowness.

In the congenital case, at least in the child, splenectomy is not so definitely indicated as in the adult. In the latter surgery is the only means of relieving the condition. Splenectomy may be considered a cure, although the method by which the relief is obtained is still somewhat obscure. We know, however, that after splenectomy abnormal destruction of red blood-cells ceases, and as a result the liver is no longer flooded with the end-products of disintegration.

Closely allied to this type of jaundice is that associated with Hanot's cirrhosis of the liver in which splenic enlargement and anemia are also found. This is possibly the final stage of Banti's disease, a name usually applied to the various conditions associated with splenomegaly and anemia, and in which splenectomy is now giving such satisfactory results. The history of Banti's disease is comparatively recent, the condition having been first pictured by its sponsor in 1882, and little more has been added to his description. Banti believed that an unknown infectious agent produced the changes, which are of a non-inflammatory character, although he was unable either to isolate the organism or produce the disease experimentally. In fact, even up to the present time, all attempts to identify the infectious agent have been futile. Various theories have been advanced with regard to the cause of splenic anemia. By some authors it is considered a functional disturbance rather than a disease of the spleen, that is, through the loss of vasomotor control of the splenic artery, caused by disease of the sympathetic ganglia, the splenic vessels become engorged, thus leading to hypertrophy and increased functional activity of the endothelial cells, which, in turn, destroy both the diseased and the healthy red blood-cells. The rapid regeneration of erythrocytes and increase of hemoglobin after the spleen has been removed indicates that not diminished blood formation but hemolysis is the cause of the anemia. It also shows that the blood-forming organs which have been so overtaxed in the attempt to counteract the rapid destruction of red blood-cells are capable of early recuperation after splenectomy. The diagnosis of Banti's disease cannot be made before the appearance of the anemia, and even then differentiation is not always an easy matter.

Pernicious anemia with marked enlargement of the spleen is not very uncommon. In these cases the anemia is apt to be more pronounced than in Banti's disease, for in the latter the hemoglobin rarely falls below 40 per cent. unless there has been a recent severe hemorrhage. The differentiation from malaria associated with anemia and splenic enlargement can usually be correctly made by the history of the case, and the presence of the plasmodium in the blood.

It is most important to differentiate Banti's disease from cirrhosis of the liver, especially since, as has already been indicated, the latter condition is often present in advanced stages of the former. We may, however, venture the diagnosis of hepatic cirrhosis when the enlargement of the spleen occurs subsequent to the change in the liver, and, of course, when the age and symptoms of the patient are typical. In the diagnosis of Banti's disease it is well to remember also that there is no necessary connection between the previous history and the development of the disorder, except that syphilis is known to affect the spleen. But neither malaria nor other infectious diseases seem to be of etiologic significance. Nor is there any apparent preference for age or sex, race or country. I have recently operated for the disease on a female child aged twenty-two months, and other surgeons report patients as old as seventy-two years. The great majority of cases, however, do occur between twenty and forty-five years of age, and cases are reported as frequently in males as in females, and in one country as well as in another.

The chief pathologic change is, of course, the splenic enlargement which progresses steadily and uniformly until the organ attains a weight anywhere from 1500 to 6200 grams. Adhesions often develop and cause technical difficulties at operation; indeed, sometimes making splenectomy impossible. There is always more or less marked sclerosis of the splenic vein and generally some change in the portal vein. In fact, some authors believe that the primary lesion is in the portal vein, the splenic changes being secondary to the congestion thus induced. The changes in the liver itself, however, are secondary. That is, they occur in the latter part of what is usually considered the second stage of the disease and the liver is then indistinguishable from Laënnec's atrophic cirrhosis.

The progress of Banti's disease is slow and chronic, enduring from three to five years or more before reaching its second stage with the anemia. It would take us too far from our present subject to discuss the blood changes on which the anemia rests. We are more interested in the third stage, the one in which jaundice often develops. The differential diagnosis from cirrhosis of the liver is a matter of nice distinction, and has already been briefly referred to.

In cirrhosis of the liver splenectomy is sometimes resorted to and has been giving satisfactory results which have been ascribed to the removal of the splenic circulation, thus diminishing a large part of the portal blood volume. In Banti's disease splenectomy seems to be the only rational treatment. In performing a splenectomy the most important thing is the control of hemorrhage. The capsule of the organ is easily torn, the pulp is soft and friable, the vessel walls in the pedicle are thin or sometimes calcareous and brittle; gentle manipulation is, therefore, essential. The manner of approach will depend upon the conditions present. If the enlargement is not too great, the pedicle can sometimes be reached from the front, after drawing the stomach well to the right and dividing the gastrosplenic omentum. The vessels in the pedicle are usually most conveniently reached by turning over the spleen, after releasing any adhesions that may be present, and dividing the splenophrenic fold of peritoneum, but these adhesions are often dense and may contain large veins, so that it is advisable to divide them between clamps, and as the spleen is gradually mobilized, to pack hot gauze in the splenic bed in order to control venous oozing. The hand is then introduced between the spleen and the diaphragm, and the former, after being drawn into the wound, is turned over and the vessels in the

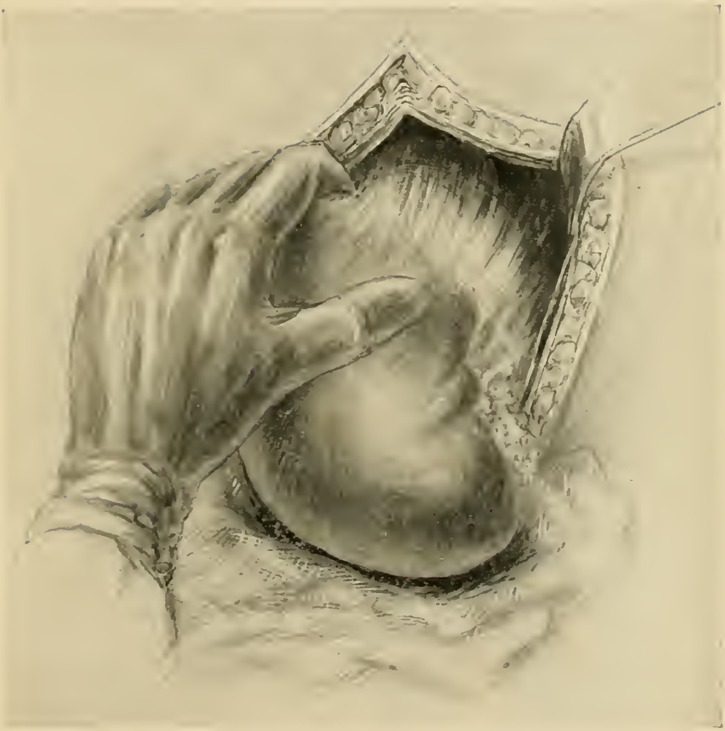

Fig. 12.—Splenectomy, illustrating the troublesome adhesions to the under surface of the diaphragm.

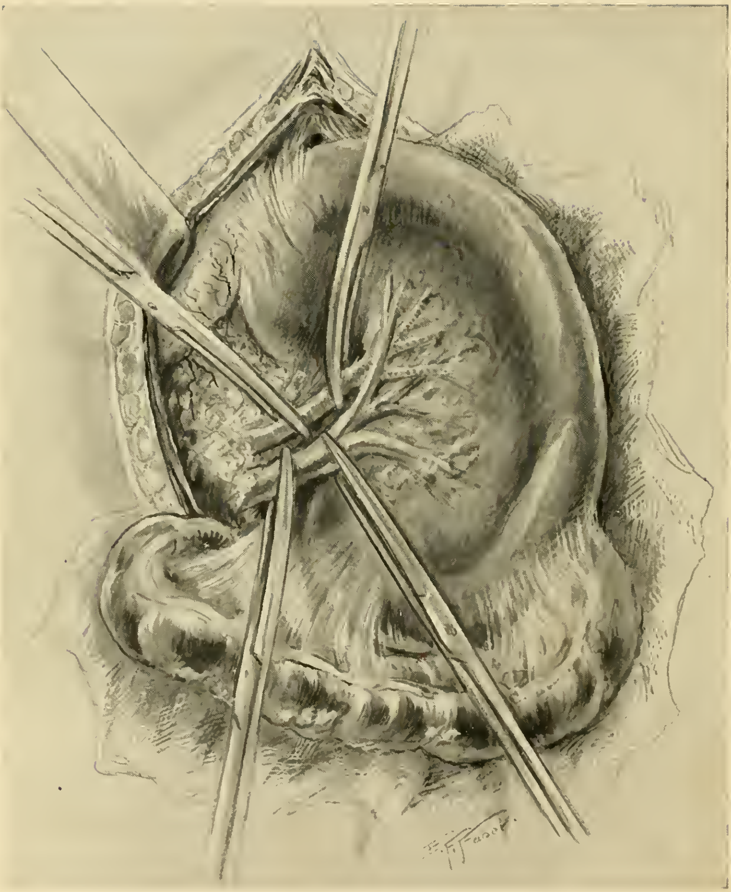

Fig. 13.—Splenectomy, application of separate clamps to the vessels.

pedicle secured between clamps and cut as close as possible to the spleen. Great care must be taken at this stage of the operation to avoid injuring the pancreas. Enough of the tissue of the pedicle must be left to permit ligation of the vessels. The ligatures should be applied separately to each of the vessels that can be identified. As soon as the bleeding from the pedicle has been controlled, the packs are removed, and after checking any bleeding in the subsplenic space the abdominal incision can be closed. If the operation is done for hemolytic icterus it cannot be considered complete without a careful exploration of the biliary passages, for in approximately 60 per cent. of cases of this disease gall-stones are present, and if the patient's condition permits, the gall-bladder should be removed. The appendix should also be removed.

Blood transfusion is often indicated both before and after splenectomy. In the chronic cases of anemia, whether of Banti's disease, hemolytic jaundice, simple splenic anemia, or pernicious anemia, where the hemoglobin is below 40 per cent., preoperative transfusion is indicated, and should be done about forty-eight hours before the operation. Of course, every careful surgeon will make a study of the cardiovascular and renal conditions before resorting to surgery in these cases. There is little doubt that to the judicious employment of blood transfusion we owe the gratifying reduction of the operative mortality of splenectomy, although the wise selection of cases and a better understanding of the technical difficulties of the operation have also contributed to the comparative safety of the procedure. Early statistics gave a mortality of about 38 per cent., while in recent reports the operative mortality is about

8 per cent. Personally, I have performed splenectomy for Banti's disease in 10 instances, and have obtained an operative recovery in all. The most serious operative complication is hemorrhage, the potential sources of which are numerous, but with careful manipulation and careful attention to control, this danger can nearly always be avoided. The postoperative sequelæ most to be feared, aside from shock and collapse due to severe hemorrhage, are bronchitis and pneumonia, which have proved the most frequent cause of death after the danger of shock has been overcome. One of the late complications, which must always be kept in mind, is intestinal obstruction due to a loop of jejunum becoming adherent to the splenic stump or to a raw area in the subsplenic space.

As to the end-results of a successful splenectomy it seems to be proven conclusively that the presence of the spleen is not essential to life; that the spleen is not the cause, but rather the agent that destroys the red blood-cells, as in hemolytic jaundice, and, furthermore, that in cirrhotic disease of the liver, removing the spleen enables the remaining portion of the liver to regenerate sufficiently to carry on the function of the organ so indispensable to metabolism.

So while we may have traveled a rather circuitous route from jaundice to splenectomy, we have, in reality, not deviated so much as at a first glance would seem evident.

DISEASES OF THE BILE-PASSAGES

From the preceding lecture it is but a natural step to consider the diseases of the bile-passages, since jaundice of the obstructive type is generally attributed to some disturbance in this region. The prime factor in surgical disease of the biliary tract is, without doubt, some form of bacterial infection. The exceptions to this statement are neoplastic disease, and the rare instances of degenerative conditions resulting from some form of toxic poisoning, such as phosphorus, arsphenamin, and the like.

Bacterial invasion of the biliary tract may give rise to inflammatory phenomena in the form of an acute or chronic cholecystitis, with or without stones, cholangititis, choledochitis, pancreatic lymphangitis, and pancreatitis. The manner in which infection reaches the bile-passages is still a matter of controversy, but the discussions of the various possible routes of bacterial invasion have been most valuable and stimulating, and have greatly influenced the surgery of the biliary tract.

Three avenues of entrance into the biliary system enter into consideration: first, by direct extension through the common duct from the intestinal tract. Until comparatively recent years this was believed to be the most likely avenue of infection; secondly, the infection may be hematogenous in origin and the bacteria may enter through the portal system or the hepatic artery; thirdly, it may enter through the lymphatics. This last avenue of entrance has only recently been receiving consideration, but evidence is accumulating to show that it is probably the most important of all. In passing, it may be mentioned that the gall-bladder may be, but rarely is, infected from an inflamed contiguous organ.

The first theory, direct extension through the common duct, loses some of its force when it is remembered that the duodenum normally is almost entirely devoid of bacteria, and that the sphincter action of the muscle of Oddi at the ampulla of Vater prevents a a constant steady flow of bile through the opening into the duodenum. It is only while food is present in the duodenum that organisms are present in that part of the intestine, and at this time the expulsion of bile is intermittent, thus clearing the opening and its immediate neighborhood of any bacteria that may possibly lodge there.

The fact that the pancreas is not a common site of infectious disease has been advanced as a strong argument against the entrance of bacteria through the ampulla of Vater, many investigators claiming that the pancreatic juice does not encourage bacterial life; nor is there any reservoir in which the pancreatic secretion may be stored. On the other hand, not only does bile stimulate certain kinds of bacterial growth, but collecting as it does in the gall-bladder, it provides a suitable lodging-place in which bacteria can develop and multiply.

But the presence of organisms in the gall-bladder bile does not necessarily mean an infection of the walls of the gall-bladder. Evarts Graham found it practically impossible to produce cholecystitis in dogs "merely by the introduction of organisms into the lumen of the normal gall-bladder, even in thick suspensions." Quoting Rolleston on this point: "In typhoid fever the bacilli

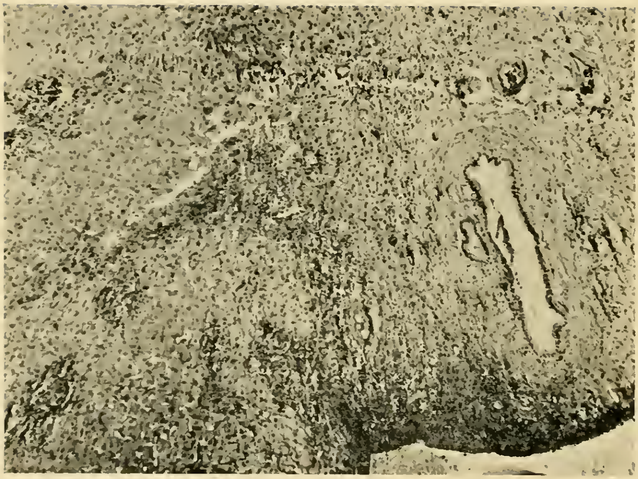

Fig. 11.—Section of liver removed adherent to a gall-bladder, the seat of a chronic calculous cholecystitis with an acute ulcerative and suppurative exacerbation superimposed. There is extreme cloudy swelling of the parenchyma, with acute and chronic inflammatory cells infiltrating the portal spaces.

are always present in the gall-bladder, but cholecystitis is comparatively infrequent." It is the mucous membrane of the gall-bladder that affords surface protection against infection.

When infection has been carried to the liver or to the gall-bladder by any of the aforementioned routes, the question arises as to the relation of these two organs and the spread of disease from one to the other. Although the natural bactericidal action of the liver cells is sufficient to overcome and destroy most of the germs carried to them, a lowered resistance of these cells either through disease or overwork may allow some of the organisms to escape destruction and be excreted with the bile, or pass into the blood-stream or the lymphatics.

The rôle of the liver cells in infections of the gall-bladder and bile-passages is also evident in the many instances in which hepatitis, in a more or less pronounced degree, is found to accompany those disorders. Dr. Reimann called my attention to this as early as 1917, but it is the experimental work and investigations of Evarts Graham and Peterman that have presented the subject to the profession.

However, it is not through the bile-ducts or the blood-stream, but by way of the lymphatics that the infection in the gall-bladder walls takes place from an infected liver. The aforementioned authors have called attention to Sudler's demonstration of the anastomotic connection between the lymphatics of the gall-bladder and of the liver, particularly extensive at the sides of the former organ. In their own experiments they have been able not only to confirm this fact but also to explain by means of this lymphatic connection the frequent association of infections of the portal system (appendicitis, typhoid fever, peptic ulcer) with lesions of the biliary tract. Furthermore, their work suggests the possibility, which I believe to be a probability, of the establishment of a vicious circle between the gall-bladder and the liver, whereby one becomes infected by the other. They show that after injecting organisms into the portal vein a hepatitis is followed by a cholecystitis, and also that hepatitis follows a cholecystitis produced by direct inoculation of organisms into the gall-bladder. This naturally establishes the rationale of cholecystectomy as against cholecystostomy in the treatment of disease of the gall-bladder, for by the radical operation the vicious circle is broken, and at the same time the disease of the liver is given an opportunity to subside. It is because of the interstitial infection of the gall-bladder in cholecystitis that I favor the more radical operation, since I do not believe an interstitial infection can be cured by mere drainage.

With the lodgment and activity of bacteria in the biliary tract we may expect the usual sequelæ of infection, varying according to the virulence of the invading micro-organism, the resistance offered by the structures affected, and the anatomy of the part of the tract involved. The disease may range from a mild, acute, short-lived infection to a violent, fulminating, and gangrenous process; or from a mild, almost symptomless catarrhal, to a chronic inflammation with acute exacerbations, each attack leading to greater and more permanent damage. The presence of inflammation in the bile-ducts or in the gall-bladder produces a thickening of the mucous membrane of the cystic duct with a narrowing of the lumen, and consequently obstruction to the flow of bile. The bile stasis, in turn, gives the organisms present a chance to exercise their full power, the resultant processes depending on their virulence and on the power of the gall-bladder and the ducts to battle against and overcome the infection. One of the possible results of this internal warfare is the development of gall-stones, the origin of which has called forth an interesting and extensive literature.

Early writers, such as Helmsback (1856), compared calculous formation in the human body to the development of pearls and shells in molluscs; that is, the stone is formed on an organic framework possibly composed of the products of inflammation, to which organic salts, such as calcium, adhere and crystallize; the definite form of the stone depending upon the pressure to which it is subjected. This same idea forms the basis of the theory of gall-stone formation advanced by Naunyn in 1892, and which is the theory generally accepted today. Naunyn believes further that the stones are usually formed within the gall-bladder as a result of catarrhal inflammation of its mucous membrane, followed by desquamation of epithelial cells and the deposit of substances normally present in bile, particularly cholesterin and bilirubin-calcium. This indicates an inflammatory origin of the bilirubin-calcium stones as well as of the cholesterin pigment stones, due primarily to the action of bacteria in causing a stasis or sluggishness in the flow of bile. On the other hand, the majority, probably about 70 per cent., of patients suffering from gall-stones are parous women, and in them the symptoms often date from pregnancy, when the bile is concentrated and stasis is present, so that the factor of stasis, which is purely mechanical, must certainly be considered. Rous and McMaster point out that stasis leads to thickening of the bile, and even in uninfected and uninflamed bile-passages this must have some effect in promoting the formation of stones, but it is around the origin of the pure cholesterin stones that most of the controversy centers. Aschoff and Bacmeister have demonstrated that the presence of pure cholesterin stones produces no inflammatory changes in the gall-bladder. According to Aschoff this type of stone develops independently of bacterial invasion as the result of bile stasis due to mechanical causes, such as stricture, pregnancy, etc., but it may be added that the irritation due to the presence of the stone may lead to secondary inflammation of the gall-bladder. These same authors have also shown that in infected gall-bladders cholesterin crystals and calculi are present in the glands of Luschka in the early stages of stone formation. The organisms surviving in these clefts or glands may account for reinfection of the gall-bladder and the recurrence of symptoms in certain cases. Other authors have shown that theoretically it is possible for pure cholesterin stone formation to take place in sterile collected bile by autolysis, but it is doubtful (as Rost points out), whether the conditions *in vitro* are analogous to those in the living human body. I have briefly alluded to these various theories mainly to indicate the trend of the controversy. Whatever may be the origin of the stones, the practical importance to be derived from the discussion is that it is not for the stone but for relief of the inflammation caused by or resulting from its presence that surgery is demanded.

Inflammation of the biliary tract may lead to a more or less mild or severe cholangitis or cholecystitis with or without the formation of calculi. Whether the infection in the liver and the ducts precedes or follows the infection in the gall-bladder cannot always be definitely determined, but infection of one without the other is rare. Generally speaking, the liver is first affected in the form of an acute hepatitis with cholangitis which usually subsides under medical treatment, but repeated attacks of acute infection may lead to chronic hepatitis and choledochitis, independent entities which, however, are frequently associated with chronic cholecystitis and cholelithiasis. It is for this reason as well as for its similarity to other conditions in the gall-bladder region that the diagnosis of hepatitis and cholangitis is not always easily made.

One of the symptoms of infection is recurring or relapsing jaundice resembling catarrhal jaundice. The liver may be slightly enlarged, but it is often difficult even for the experienced surgeon to detect this. Kehr states that the liver is enlarged in 15 per cent.; Graham finds it in 87 per cent. of the cases, while Judd, of the Mayo Clinic, reports hepatic enlargement as rather infrequent. In the absence of infection of the bile-passages the disease runs a mild course, with remissions in the intensity of the jaundice, but, as I have already mentioned, it is rather difficult to tell whether the jaundice is due to obstruction of the common duct, to a choledochitis, or possibly to a pancreatitis secondarily obstructing the bile-passages, or perhaps to malignancy. Cases of prolonged jaundice due to cholangitis, therefore, should be subjected to an exploratory operation in order to ascertain and treat the underlying or associated condition by draining the gall-bladder or the common duct according to indications. As the result of persistent infection of the bile-passages, we naturally have cholecystitis unaccompanied by gall-stones in only about 20 per cent. of cases. The symptoms of non-calculous cholecystitis are much like those of the calculous disease, and in both it is the degree of infection and the resultant inflammation that dominates the picture and affects the prognosis. Chronic non-calculous cholecystitis bears the same relation to the upper abdomen as does chronic appendicitis to the lower abdomen. It is a much more frequent pathologic entity than has hitherto been believed. To me it represents the forerunner of the relatively more dreaded calculous cholecystitis.

As to cases of acute cholecystitis, a first attack may subside under expectant treatment, such as complete physiologic rest, cold applications to the abdomen, and the infusion of fluids by proctoclysis, but as far as I am able to judge, the so-called non-surgical drainage of the gall-bladder by means of magnesium sulphate introduced directly into the duodenal lumen through a duodenal tube, in order to cause relaxation of the sphincter of Oddi, can have no influence whatever on an acutely inflamed gall-bladder and cystic duct. This same method is also being advocated as a means of diagnosing cholecystic disease. The action of the magnesium sulphate is supposed to cause contraction of the gall-bladder and relaxation of the sphincter of Oddi, thus allowing the bile to flow freely into the duodenum. In this way the bile coming from the common duct, that from the gall-bladder, and that from the liver are said to be differentiated, and from an examination of each type the existing conditions are to be determined. Personally, I have not found the method of great help, since the condition of the bile does not always indicate the existing pathology in the gall-bladder and the liver ducts. The method, however, is finding favor with many clinicians, and for that reason deserves further study.

The damage done even by a single attack of acute cholecystitis, even though the lesion be a slight one, is apt to persist, so that with each recurrent attack the lesion naturally is aggravated. Moreover, experience and experiment have taught us that these lesions rarely remain confined to any one viscus. The association of cholecystitis with duodenal ulcer and appendicitis is too common an occurrence to be merely incidental.

From the symptoms of cholecystitis one cannot very well tell whether or not stones are present, so that the clinical picture of the calculous and the non-calculous conditions may well be considered together. They occur chiefly during and after middle life, rarely in childhood; but we must not forget that they may be seen at any age from the fifteenth to the eightieth year. I have already said that the calculous disease is much more frequent in women than in men, the ratio being about 4 to 1. Although the ratio in the non-calculous disease may not be quite so high, it is also definitely in favor of the female sex. We have found that our cases when analyzed according to symptoms can be made to fall into four groups as suggested by Jacobson:

1. *Typical biliary colic group.* These patients give a history of recurrent attacks of pain and tenderness in the right hypochondrium or epigastrium. The pain is sudden in onset, coming on at any time, but more often at night. It rapidly increases in severity and frequently radiates to the right, affecting the back, the shoulder, the neck, or the arm, and requiring morphin for relief. Nausea and vomiting are usually present. Vomiting is not a constant symptom, but when present the vom-

itus is apt to be bile stained. The acute case is accompanied by chills and fever, frequently followed by jaundice. This jaundice may be so transient and slight as not to be recognized when it does occur. The scleræ may be faintly tinged, and perhaps only a trace of bile will be found at the chemical examination of the urine. The attacks usually subside in a few hours, but tenderness in the gall-bladder region may persist for several days. The history is usually a prolonged one, extending over several years, with the attacks increasing in severity and frequency as time goes on. At first the intervals between the attacks are apparently free from symptoms, but gradually more or less marked indigestion develops between the acute attacks, the outstanding symptom being belching of gas and distention after meals. As you probably know, my favorite dictum is, "fair, fat, and forty, belching gas, equals gallstones." However, this period of freedom from symptoms between attacks does not mean a cure of the disease, nor does it mean an entire latency of symptoms. One of my colleagues has well said that "innocent gallstones are a myth."

2. The second group may be classified as the *typical biliary group.* These cases do not have attacks of acute biliary colic. The patients complain of a dull ache in the right hypochondrium associated with tenderness in the same region. It is frequently difficult to elicit tenderness in this group, but by careful examination a more circumscribed area can usually be noted than that in which the pain is felt. Although the pain may be continuous, the majority of the cases have periods of remission. Fever, nausea, and vomiting accompany the attacks, while jaundice is an inconstant feature.

3. The third comprises *the gastric group.* The digestive symptoms, as well as the clinical picture, often resemble those of peptic ulcer. The points of distinction are mainly in the relief of pain obtained by food or alkalies in the case of ulcer, and also in the fact that in the latter there is no relation between the type of food and the pain. In other words, the ulcer patient complains of pain no matter what he eats ("quantitative" food dyspepsia), while the gall-bladder patient usually finds that certain foods bring on his digestive troubles ("qualitative" food dyspepsia). These cases are frequently treated for years for "gastric catarrh," gastric or duodenal ulcer, or merely for dyspepsia, without obtaining permanent relief. They are the cases which need the most careful study in the shape of analysis, radiograms, and examination of urine and feces. The recognition that the gall-bladder may reflexly simulate gastric pathology is only one of the many results of our observations on "living pathology."

4. Finally, we come to the fourth, *the biliary-gastric group*, which combines the symptoms of the first and third groups. It may begin as group one and later have added to it the symptoms of group three, or vice versa. There is apparently no constancy in its clinical picture.

Diagnosis of disease of the gall-bladder oftentimes is apt to be confused because of the presence of an inflamed appendix in a high position. I freely confess that clinical differentiation between this type of appendicitis and disease of the gall-bladder is often impossible. Chronic appendicitis also is so frequently associated with biliary tract disease that no operation in this region is complete without an associated appendectomy. I speak from personal experience of many such instances.

As for Roentgen-ray diagnosis, I think I am not alone in finding it more reliable as confirmatory evidence than in the detection of disease of the gall-bladder itself even when stones are present, particularly since not all kinds of calculi throw a shadow in the roentgenogram. The usual test-meals and gastric analyses are sometimes of value, but even these fail to assist in the differential diagnosis between high appendicitis and cholecystitis. Early cases of mild cholecystitis usually have a hyperacidity, but in the later stages of chronic gall-bladder disease we are much more apt to find a hypo-acidity or, even in certain cases, anacidity. The final verdict can be obtained only from ocular evidence at the operating-table.

The two important questions that confront the surgeon are, when to operate and what operation to choose. As to the first, it may generally be said that in the chronic cases the time may be chosen at the convenience of those concerned, but it must be borne in mind that the end-results are favorable in direct proportion to the duration of the disease. In other words, the greater delay in removing the focus of infection, the less likelihood will there be of a complete return to normal health. The dangers and discomforts to which patients expose themselves when they select palliative treatment are: (1) Persistence or recurrence of the pain; (2) inflammatory complications, ending in adhesions, ulceration, and gangrene, often necessitating an immediate emergency operation; (3) the occurrence of pancreatitis; (4) the development of cancer.

Operation during an acute attack of cholecystitis without jaundice is a more delicate question. With the usual regulation treatment, anatomic and physiologic rest, the attack may subside sufficiently to make operation safe, but the danger of jaundice or rupture of the gall-bladder and the extension of the disease to the pancreas must enter into consideration. The extent of peritonitis present also must decide the wisdom of immediate interference as against delayed operation.

The presence of jaundice naturally presents a still more delicate question as to the best time for surgical intervention. In cases where the jaundice is in its incipient stage immediate operation may be undertaken with considerable safety, but in the presence of deep jaundice which shows signs of subsiding it is probably safer to wait until it has diminished as far as it is likely to without removal of the underlying cause. The danger to these patients lies, of course, in hemorrhage. While the coagulation time of the blood may at times indicate the degree of risk, it does not always do so. Fortunately in blood transfusion we have a means not only of preparing jaundiced patients for operation and lessening the risk of hemorrhage, but also a useful postoperative measure for restoring the coagulation time to normal and preventing postoperative bleeding. The work of Lee and Vincent and of Whipple on the intravenous use of calcium chlorid is a most valuable contribution to this subject. Whether it is used in small amounts of higher concentration, as suggested by Lee and Vincent, or in large amounts of a weaker concentration, the coagulation time can be reduced and the accompanying toxicity diminished.

As to the type of operation: In the early days of gall-bladder surgery we heard much of the relative merits of cholecystostomy versus cholecystectomy. With the development of, and acquired proficiency in, the technic the question of the relative safety of the two procedures became unimportant, since the difference in immediate mortality between the two was found to be minimal.

On the other hand, studies of the final results of operation showed that recurrence of symptoms was much more likely after the conservative cholecystostomy than after the radical operation of cholecystectomy (the proportions being about 70 to 30 per cent.). In passing it may be said that recurrence takes place, irrespective of the operation, in about 4 per cent. of the cases. In most instances I find that the return of symptoms after cholecystectomy is due to the development of adhesions binding the duodenum and possibly also the pylorus, the great omentum, and sometimes the hepatic flexure of the colon to the under surface of the liver. This postoperative sequel can in many cases be avoided by careful technic during the removal of the organ and also by interposing a portion of the great omentum between the under surface of the liver and the duodenum at the final stage of the operation.

After the drainage operation the commonest cause of the return of symptoms is re-formation of stones in the gall-bladder. This of itself speaks in favor of removal of the organ, if at all possible. At the same time in some few instances even after cholecystectomy stone formation takes place in the common duct or in the hepatic duct. Calculi in the hepatic duct may be new stones, or they may have been present at the original operation and have been overlooked or, more likely, it may have been impossible to remove them at that

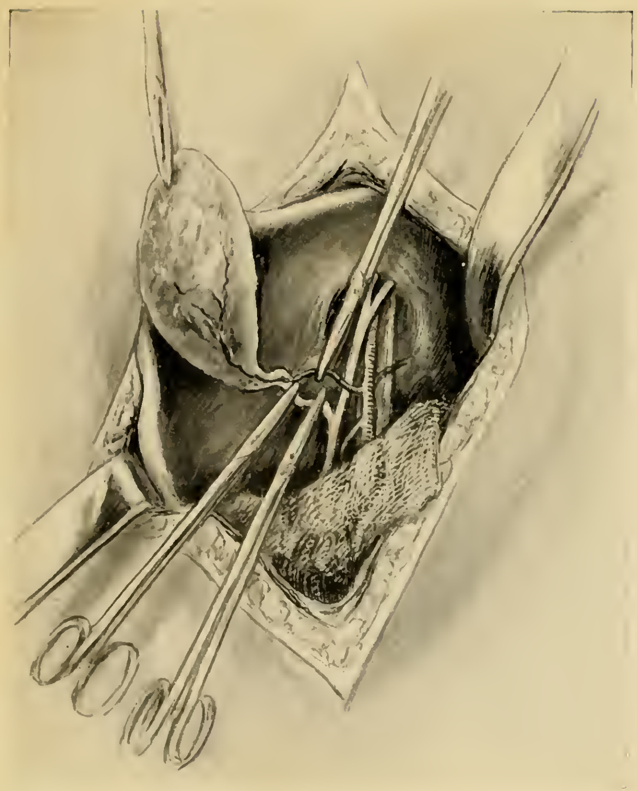

Fig. 15.—Separate clamps placed on the cystic artery and the cystic duct.

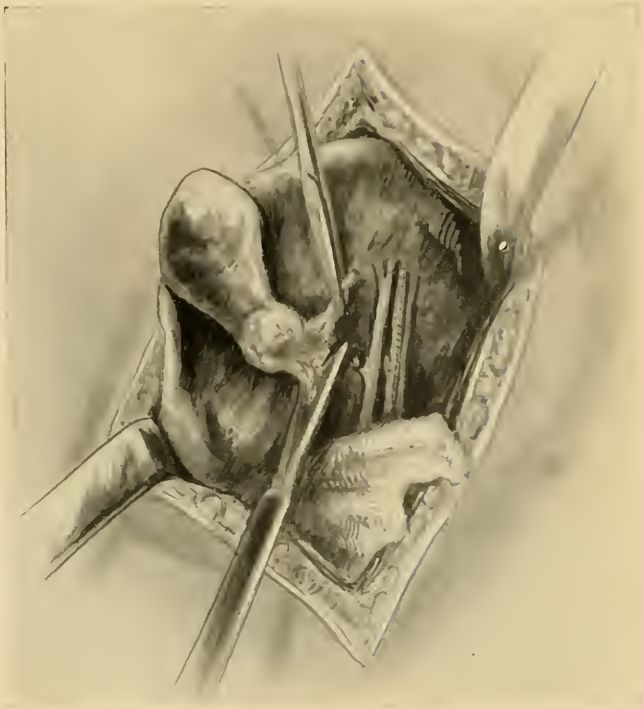

Fig. 16.—The removal of a gall-bladder containing a large stone impacted in its neck.

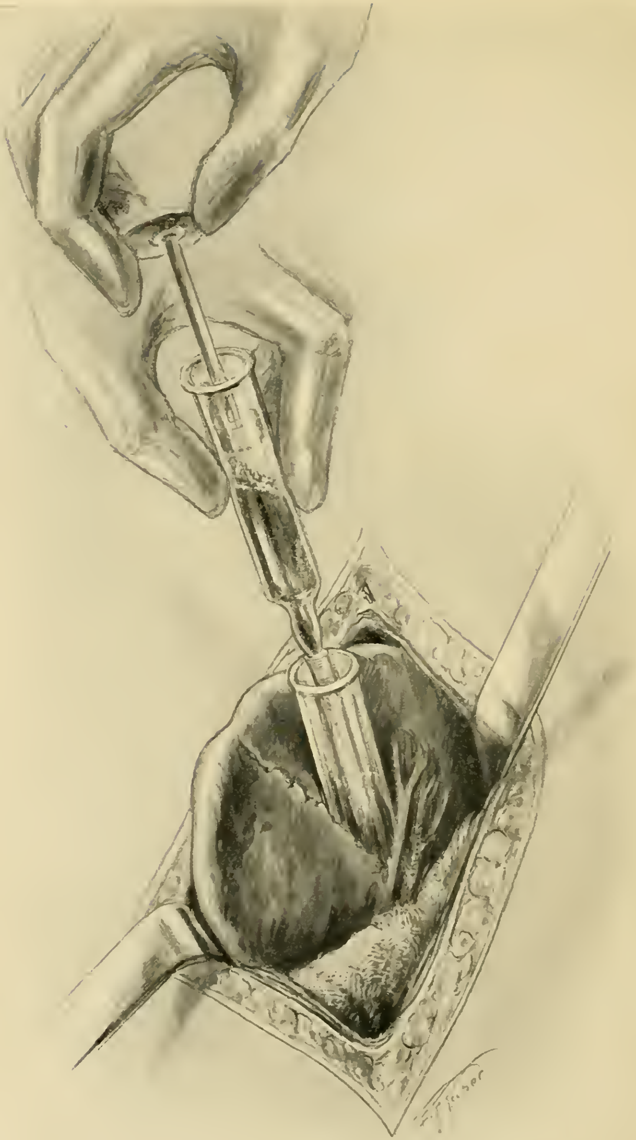

Fig. 17.—The mode of removal of the material collected in the subhepatic fossa through a glass tube. This illustrates the technic at operation, but the same maneuver is used postoperatively.

time. A striking feature of many of the cases of recurrence of cholecystic symptoms after operation is their latency. While in a small number of instances the patients begin to experience symptoms within a few weeks or perhaps several months after operation, a considerable number go through a period of well-being extending over two or three or even as many as ten years before the symptoms reappear. Whether these are actual recurrences in the shape of a flare-up of a latent, persistent infection, or a renewed infection is a difficult matter to decide.

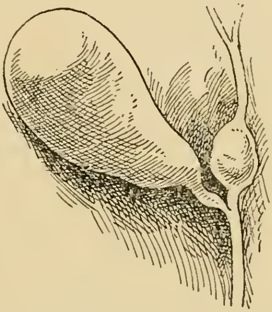

Fig. 18.—Stone in the hepatic duct.

In a few instances the recurrence of symptoms is traceable to a pancreatitis or to a hepatitis, or to both. Judd has recently called attention to this type of case, and suggests that the pancreatitis and the hepatitis may have been present at the primary operation and that by draining the common duct, the rational treatment for pancreatitis, the return of attacks after removal of the gall-bladder might have been avoided. The close relationship between cholecystitis and pericholecystic disease and pancreatitis is a well-established fact. Linder states that 50 per cent. of the cases of pancreatitis depend upon or follow disease of the gall-bladder with gall-stones. Jacobson quotes the Mayos as reporting that 85 per cent. of all pancreatitis cases are associated with gall-stones, and 25 per cent. of them are common duct stones. In Jacobson's series of 397 gall-bladder operations, acute and chronic pancreatitis was found in 14 instances, 5 of them being of the acute hemorrhagic type. It is becoming more and more recognized at this time that infection is carried from the gall-bladder to the pancreas by way of the lymphatics or through the reflux of bile into the pancreas because of obstruction at the ampulla of Vater, and the resulting irritation or infection, if the bile is septic, should be considered. The lymphatic system of the pancreas is known to be a rich one. The organ being without a hilus, the lymphatics, like the blood-vessels, emerge along the surface and run along the gland to neighboring trunks or plexuses, sending branches to anastomose in the cellular retroperitoneal tissues with lymphatics from the stomach, the duodenum, the spleen, the liver, the gall-bladder, the bile-ducts, the colon, and even the left suprarenal body. But the communication is particularly free between the lymphatics of the duodenum and the head of the pancreas and between the gall-bladder, the bile-ducts, and the pancreas. Pancreatitis unassociated with pathology in the neighboring viscera is not common, so that the conclusion is evident that the organ becomes affected as the result of disease, usually infection, of one or the other of its neighbors. While in some instances the pancreas is involved in a primary affection of the stomach, the duodenum, and occasionally the appendix, it is from disease of the biliary system, particularly cholelithiasis, that it most frequently becomes involved. The sequence

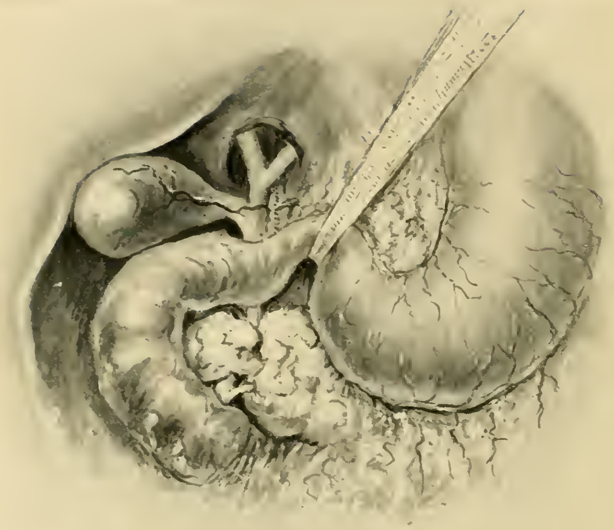

Fig. 19.—The pylorus is pulled aside to show sclerosis of the head of the pancreas.

of events as seen at the operating-table is about as follows: Hepatitis, cholecystitis, lymphadenitis in the gastrohepatic omentum, and along the course of the cystic and of the common duct, peripancreatic lymphadenitis, pancreatic lymphangitis, chronic pancreatitis, and occasionally acute pancreatitis.

Pancreatic lymphangitis is a distinct clinical entity, but it has not been recognized as yet because the lesions in the pancreas itself are slight, inasmuch as the organ is still able to supply both its internal and external secretions to the body economy and because the localizing symptoms of inflammation, such as pain and tenderness, are so intimately connected with these signs in the diseased gall-bladder as to be indistinguishable from them. The pancreas, however, is undoubtedly affected, especially in cases of long-standing ailment with symptoms of common duct involvement and tenderness over the head of the organ. This peripancreatic and pancreatic inflammation is very much like the adenopathy which follows infection elsewhere in the body, such as cervical adenitis secondary to tonsillitis, and inguinal adenitis after infectious lesions of the perineum. In its early stages this pancreatic lymphangitis subsides with subsidence of the infection in the gall-bladder. But long-continued lymph stasis and infiltration naturally lead to destruction of the parenchyma and the deposit of fibrous tissue. From this to swelling of the pancreas and thence to obstruction of the ducts is but a matter of time. In some instances I have found an abscess in the pancreas, and it is not beyond the range of possibility that certain cases of hemorrhagic pancreatitis may originate from infection of the neighboring viscera by way of the lymph-channels. That hemorrhagic pancreatitis does not always arise from impaction of a gallstone in the papilla of Vater, the generally accepted cause of the disorder, is proved by the fact that quite a number of cases are not accompanied by the presence of calculi. The frequent association of pancreatitis with cholecystitis again emphasizes the importance of early removal of the infected focus and the evils of procrastination. It is also another argument in favor of cholecystectomy rather than cholecystostomy, first, because the infected tissue is removed and, secondly, because the dilatation of the common duct following removal of the gall-bladder allows for adequate drainage.

Pathology

In discussing diseases of the gall-bladder and biliary passages from the laboratory standpoint it is probably better to consider first the physiology, then the pathology, and then the bacteriology, and finally certain points which may be deduced from consideration of those facts. The gall-bladder, we believe, has two main functions—(1) concentrating, (2) propulsive. If we examine the first property we will find that it is only within the last several years that this function has been estimated quantitatively. It had been known, of course, for many years that the bile from the gall-bladder is very much richer than that obtained directly from the liver. Numbers of analyses showing this difference have been made, but the rate and the extent to which the gall-bladder could concentrate the bile was not known until the work of Rous and McMaster. It has been found by an ingenious method of collecting bile directly from a part of the liver into a rubber bag and comparing it with the bile coming from another part of the liver, but which has been allowed to remain in the gall-bladder for a certain length of time, that in twenty-four hours the bile may be concentrated as much as eight times. We know, furthermore, that bile does not flow continuously into the duodenum, but that the opening of the common duct into that viscus is controlled by a sphincter muscle named after Oddi. There is some information at hand concerning when and under what conditions the sphincter opens to allow the entrance of bile into the duodenum. Bile is necessary in the intestine during the digestion of food, but there are serious gaps in our knowledge of the time and the conditions under which this sphincter opens. In other words, there is no unanimity of opinion concerning the influence of various kinds of foods on the opening and closing of this sphincter. However, the liver secretes bile in amounts greater than could be contained within the gall-bladder and the bile-ducts unless some special mechanism were present to provide storage space. This the gall-bladder does, not by reason of its large storage capacity, but by means of concentrating the bile as it enters. One might say, of course, that gall-bladder bile is much more of an aid to digestion than liver bile, since it contains the materials in a more concentrated form. It is very interesting, especially from a teleologic standpoint, that Rous and McMaster find that the bile of the rat, an animal without a gall-bladder, and hence one which has no concentrating organ, contains eight times as much pigment as does the liver bile of the mouse, which has a gall-bladder.

The next question concerns the method by which the fluid, as it is withdrawn from liver bile, is conveyed from the gall-bladder. I know of no definite answer to that in the literature as yet, but from data which we have, we can at least say that considerable fluid is carried off by the way of the lymphatics. This conclusion was arrived at by passing a small catheter into the gall-bladder through the papilla of Vater, after having first carefully clamped with mosquito forceps the ducts from the various lobes of the liver. The gall-bladder was then washed of its content of bile and filled with warm saline solution under various degress of pressure as controlled by manometers. Under favorable conditions, that is, in an animal (dog) the ducts of which allow of clamping without undue obstruction of surrounding vessels, it has been found that saline disappears oftentimes at the rate of 1 c.c. in four to five minutes. Coincidently, the lymphatics in the walls of the gall-bladder shining under and in the peritoneum become conspicuous, and a number at the neck become quite dilated, especially one larger vessel which is in close apposition to the cystic artery. If this larger vessel is nicked with scissors the whole field of observation becomes flooded with fluid, which re-accumulates at a rapid rate after being mopped out. That some of this fluid comes from within the gall-bladder can be shown by adding a soluble dye, such as phenolsulphonephthalein, to the saline which is introduced into the gall-bladder. We do not know how much has directly entered the blood-stream, possibly some. If this procedure is prolonged for several hours, the tissues in the gastrohepatic omentum as well as the gall-bladder walls become highly edematous. The drainage is seen to take place in a number of directions. We were especially interested in that occurring toward the region of the head of the pancreas, and found that a part of the current was directed into this locality. A modification of this procedure in which dogs and rabbits both were used, by which India-ink was placed in the gall-bladder, with or without occlusion of the cystic duct, but with obstruction to the common duct at the sphincter, showed that the granules spread in all directions, some reaching the region of the pancreas as well as the bed of the gall-bladder, some even appearing on the dome of the liver after twenty-four to thirty-six hours. In the latter experiments, of course, the operation was performed under strict asepsis with tight closure of the abdominal wound.

The next point for consideration is the second function, the propulsive activity of the gall-bladder. Smooth muscle can be found in the gall-bladder as well as in the bile-passages, but it is very small in amount, and, of course, less in the dog than in the human subject. It has been assumed that when the papilla of Vater opens, the gall-bladder contracts and projects its contents downward into the duodenum, but no such mechanism has ever been demonstrated. One would say *a priori*, that with the small amount of muscle present, very little force could be exerted. If we examine the records of the various experimental procedures intended to determine the propulsive power of the gall-bladder we will find one feature in common, that is, that the gall-bladder possesses very little or no such power. It may not be out of place to mention a few of the procedures which have been used to study this problem. Most of those designed to record contractions graphically are beset with the difficulties of distinguishing any movements of the gall-bladder from those incident to respiration. In our own experiments this has been a most baffling factor. We have connected the fundus of the gall-bladder to a lever by means of a stitch; we have opened the fundus through a small incision and put in balloons attached to tambours; we have succeeded in passing a rubber balloon into the gall-bladder up through the common duct, and then connecting it with a tambour; we have distended it with saline solution connected to a suitable manometer, and so on. By attaching strips of the excised gall-bladder, or the entire organ between levers, it is possible to demonstrate only a slow, regular, rhythmic contraction and relaxation representative of ordinary smooth muscle tonus. No better results were obtained following stimulation of all sorts applied both directly and indirectly. When we turned our attention to the sphincter of Oddi we found that after a preliminary spasm following laparotomy and incision of the duodenum, the sphincter relaxed and contracted at varying intervals. The time length of both the preliminary spasm and the subsequent contractions and relaxations varied considerably in individual animals. If the abdominal incision was allowed to remain open, bile simply ran out when the sphincter relaxed. Flooding of the sphincter region with solutions of various strengths of magnesium sulphate, sodium sulphate, sodium chlorid, peptones, fats, carbohydrates, and products of digestion from the stomachs of other dogs, etc., led to no definite results. The sphincter either opened or not, in a way which we have not been able to correlate. Periods of contraction varied from five to forty-five minutes or even longer; periods of relaxation varied from one-half minute to four or five minutes. Direct mechanical stimulation, such as touching the opening, usually led to immediate contraction. These experiments were all carried out on dogs anesthetized with ether to a depth not sufficient to abolish such reflexes as intestinal peristalsis or response when the intestine or stomach was pinched or otherwise stimulated. If, however, the duodenum was opened opposite the sphincter, and gently brought up into the wound, which was then closed tightly around it, the flow of bile, when the sphincter opened, had more force, that is, when the sphincter opened, the bile did not simply flow out as when the abdominal incision was allowed to remain open, but was forced out in little jets coincident with inspiration, as the liver descended and intra-abdominal pressure increased. When India-ink had previously been placed in the gall-bladder through a needle puncture in the fundus, the granules sometimes appeared at the papilla of Vater when the sphincter relaxed, and sometimes not. Briefly, our theory of how the bile enters the intestine irrespective of the stimuli which caused the sphincter to open is this: The sphincter closes and the bile is held under a pressure which may reach 200 or more millimeters of water. The gall-bladder concentrates the bile and so relieves some of the pressure, but the pressure is constantly higher than in the duodenum. When the sphincter opens bile runs out because of the pressure under which it is confined and because of the movements of respiration, and possibly because, as Sweet suggests, of the "milking" action of duodenal peristalsis. Forced outflow of bile from contractions of the gall-bladder does not seem to occur.

Furthermore, when obstruction is offered to the outlet of a hollow contractile viscus, the first change is one of hypertrophy. We have never seen a gall-bladder with an hypertrophied muscle layer, although we have examined a number in which stones were present in the cystic duct—an ideal condition for such an hypertrophy to occur. The pain in biliary colic when a stone is descending must, therefore, be due not so much to contraction of musculature in the attempt to force the stone outward, as to distention behind the duct, which when it reaches a certain degree will allow the stone to pass onward a small distance as bile runs around it; then obstruction occurs again with distention; then the stone passes a short distance further, and so on.

In the pathology of disease of the gall-bladder the two main considerations are inflammation and tumors, the first being of greater frequency and importance. It is true that tumors of the gall-bladder and bile-passages are often enough encountered, but from the patient's viewpoint, since they are mostly malignant, the outlook is well-nigh hopeless, so that the subject of inflammation is of far more interest. Dr. Deaver has touched upon the routes by which infection may reach the organ, He has also called attention to the digestive disturbances which are present in a diseased gall-bladder. Such studies of the visceral nervous system as are being made by Carlson and his associates demonstrate the nervous relations leading to such things as pylorospasm, etc., but if we turn to the pathologic anatomy we will find that the inflammations as they effect the gall-bladder may, by means of adhesions, bands, etc., directly involve contiguous structures. From a study of selected specimens it is possible to reconstruct a more or less complete picture of what occurs in advancing disease of the gall-bladder. The first changes are referable to the mucosa and consist of cloudy swelling, mucoid degeneration, and subsequent fatty and lipoid changes. The walls become congested, edematous, and infiltrated by inflammatory cells, the mucosa desquam-

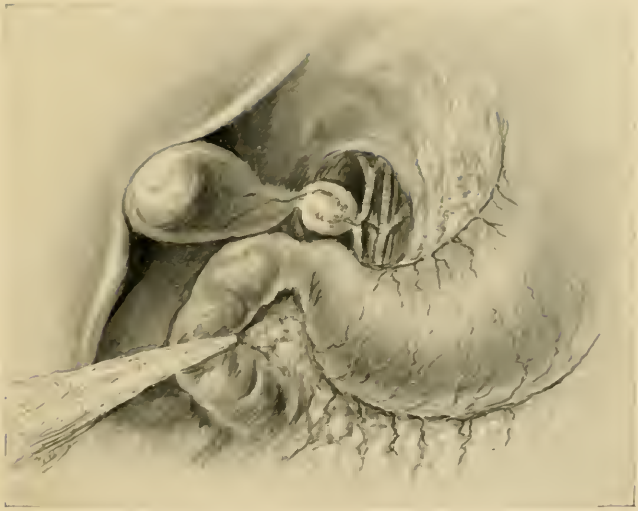

Fig. 20.—Stone impacted in the cystic duct.

ates and ulcerates. If the infection is more virulent, the entire mucosa may be destroyed, the walls of the organ become much thickened by inflammatory exudate, flakes of fibrin appear on the peritoneal surface, small areas of the wall soften, abscesses form, and finally gangrene supervenes. Perforation is the natural consequence. The content of the gall-bladder is a thick, grumous, reddish-yellow, purulent fluid. Where the infection is less virulent and is overcome before gangrene and perforation supervene, organization tissue makes its appearance. As the process abates, the organization tissue becomes connective tissue; the walls of the organ lose their elasticity and become opaque and lusterless, while the mucosa is replaced more or less completely, but with absence of the typical villous-like projections. Not all infections of the gall-bladder, however, follow such a course, in fact, most of them are of lower degree.

In the gall-bladder we are not confronted, as in the appendix, with the deposit of connective tissue due to advancing age; and when connective-tissue changes are found in the gall-bladder they are in all probability due to either one of two things. A slowly progressing chronic inflammatory process which may, of course, have ceased, but was progressive at one time or another, or the scars of previous acute inflammations. Even though the removal of the gall-bladder is not only compatible with the life of the patient but also with his general good health, and its absence under proper conditions is not missed, it still has very much more function, as far as we know, than the appendix. Actually, we do not remember having seen a gall-bladder in an older person in which connective tissue had been deposited and in which it could be interpreted as due to ordinary sclerosis without symptoms referable to the upper abdomen. The first proposition of chronic inflammation as well as the second—acute attacks—requires considerable investigation, that is, whether an infection really ceases either in the chronic progressing variety, or in between the acute attacks. Clinically, as Dr. Deaver has so often emphasized, a gall-bladder once infected is hardly ever again innocent, and thus evidences of infection can always be found in such an organ. This, of course, does not exclude patients who may have had one or two attacks of cholecystitis, or even a slight chronic progressive infection of short duration, with complete recovery. Such patients, in the first place, if they are not operated upon during the course of their disease, are not operated upon later, and so a considerable number of gall-bladders are not made available for study. Furthermore, the difficulties of diagnosis in the upper right quadrant of the abdomen are so many that the diagnosis of acute or chronic gall-bladder disease with subsequent complete recovery is very likely to have been a mistaken one. In all probability all patients who have the typical text-book gall-bladder symptoms retain an infected gall-bladder with subsequent symptoms indefinitely, or until it is removed. There must be a reason for this continued infection. The normal anatomy of the organ teaches us that there are many hiding places for bacteria in the mucosa; there are the small crypts between the villi, not to speak of the deeper hiding places in Luschka's glands, etc.; more than that, and this is the most important point on which we must lay stress, in by far the larger majority of infected gall-bladders evidence of inflammation is found not only in the mucosa but also in the interstitial tissues. In the case of the acute variety the walls become thickened and infiltrated throughout with acute exudate. In the chronic variety there is a thickening of the wall from deposits of connective tissue and further evidence of continued infection in the finding of abundant lymphoid cells—the smaller round-cells of chronic inflammation—sometimes diffusely scattered, sometimes in foci, oftentimes in streaks, no doubt following the lymphatic channels through the wall. This interstitial inflammation, we must insist, is of far greater importance than a catarrhal or other inflammation involving only the mucosa. Interstitial inflammation extending through or into the serosa gives a clear explanation of why toxins are absorbed from diseased gall-bladders; why there are adhesions in its vicinity, etc. If, then, the anatomic make-up and the pathologico-anatomic picture of the gall-bladder favors hiding places—breeding places, if you will—for bacteria, are there any studies which demonstrate that bacteria actually are present within the walls of the gall-bladder? This question has been answered quite satisfactorily. Rosenow, by his improved methods of culturing organs, has succeeded in isolating streptococci in a number of these gall-bladders. Others, including ourselves, have confirmed these results, but there are a number of interesting points which may be mentioned. In our clinic it is a routine practice to culture the contents of the gall-bladder. Taking a series of cases with no attempt at choice, the results of these cultures can be expressed as follows: Approximately half of the specimens show a growth in the bile removed from the gall-bladder either at operation or more often a few moments after cholecystectomy. The most frequent organism recovered was the colon bacillus, although we have isolated Bacillus typhosus and B. paratyphosus as well as other members of the typhoid-colon group. The streptococcus was also isolated in a number of cases, by far the majority being of the viridans type. For purposes of special study 45 gall-bladders were treated by Rosenow's method and the cultures examined with special reference to streptococci. In 20 per cent. these organisms, all of the viridans type, were recovered (in Brown's series of cases the streptococcus was isolated in 50 per cent.). The subsequent histologic examination showed that they all had a marked interstitial involvement. Other organisms were also encountered both in these and in other gall-bladders, but their identification was not carried out to a degree further than that they were probably colon bacilli. It requires no further discussion than a mere mention of the well-known ability of streptococci to remain living for prolonged periods of time with or without acute exacerbations of virulence, to harmonize with our views concerning the fact that a gall-bladder once diseased will, in the large majority of instances, either make its presence known again, usually in a more unfavorable way, or will make its presence felt constantly. Parenthetically, it might be remarked that we have recovered pneumococci from several cases. Ordinarily, bile will dissolve pneumococci, but not all gall-bladders are filled with bile, some are filled with purulent fluid containing a minimum of bile. Others are filled with a bile which is very probably weakly solvent for pneumococci; at least, in working with pneumococcus cultures, it has been our experience that not every bile collected by us (usually human bile), either from removed gall-bladders or obtained from a cholecystostomy, will dissolve all cultures of pneumococci. It is not beyond the range of possibility that certain pneumococci under conditions of being in the gall-bladder walls might acquire a tolerance to the bacteriolytic activity of bile. To sum up, therefore, we have anatomic peculiarities which favor long-continued infection, pathologic anatomic pictures which show chronic inflammation in the walls, and bacteriologic studies which demonstrate the organism. With, let us say, streptococci in the gall-bladder a chronic toxemia may be present for a long period of time; there is a constant stimulus to lymphangitis and connective-tissue proliferation, a constant menace of acute exacerbation due either to increased virulence or reduced resistance of the patient, and the danger of general bacteremia. In the particular patient who has harbored such a diseased gall-bladder it is difficult, often quite impossible, to evaluate the direct symptoms of which the patient complains at the time of operation with the infection in his particular gall-bladder. The records in our follow-up system show very interesting analytic results. Not all recover health completely after the removal of an infected gall-bladder, but we will speak of this later. The immediate point is that the symptoms at the time of operation may, and probably are, due not only to the infected gall-bladder itself, but to complications secondary to its long presence. Such conditions as adhesions, pancreatic, lymphangitis, chronic pancreatitis, and infection of the liver may be mentioned. Dr. Deaver has called attention to the work of Graham and Peterman, of St. Louis, on the lymphatic connections between the gall-bladder and liver. We have noted for quite a few years, in bits of liver tissue attached to removed gall-bladders, or bits that have been removed especially for study, that the liver in many such instances is far from normal. Briefly, the changes parallel the intensity of infection in the gall-bladder to a remarkable degree. Of course, it is well known that a cholangitis produces liver changes, but we will not speak so much of that, since it is well known, but rather of those specimens in which tiny bile-ducts in the removed liver tissue are found in approximately normal anatomic condition. In these, as well as in others, the liver cells show changes varying from mild cloudy swelling to pronounced fatty degeneration and infiltration. Kupfer's cells may be swollen to quite a marked degree, judging from the size of the bile-duct, the hepatic artery, and the portal vein in a portal space, there are also many lymphocytes; further than that, the perivascular lymphatics are sometimes dilated and contain inflammatory cells. In the more acute infections there are pus-cells in the portal spaces and sometimes also in these perivascular lymphatic spaces. Surgeons have sometimes attempted to judge the amount of enlargement of the liver clinically at the operating-table; of course, they disagree. The best and only certain method is to weigh the organ, which obviously cannot be done, but we may presume that a certain number are enlarged even though the opinion as to how many is not uniform. There are changes far more important than increase in size, and the eye trained in pathology can very readily detect at the operating-table such conditions as marked cloudy swelling, fatty changes, connective tissue increase, etc. It is reasonable to suppose that acute infections of the gall-bladder will lead to degenerative changes in the liver. Furthermore, such patients undoubtedly have degenerative lesions of the heart, kidneys, and other parenchymatous organs. The kidney condition can, of course, be found by examination of the urine, the abnormalities of which often clear up in three to four weeks after the removal of a severely infected gall-bladder.

It may not be inappropriate at this point to call attention to this phase of the subject in a more general way. Dr. Deaver has been a very ardent champion of what is called "living pathology," *i. e.*, observation of what the surgeon finds at the operating-table and subsequent study of the removed pathologic material. There is no doubt that many questions in intra-abdominal pathology have been answered in this way, and there is no doubt that this pathology is extremely important and enlightening to the general pathologist and the clinician trained in pathology, but to the medical student or to the clinician, whether surgeon or internist, who has not been trained in general pathology or has forgotten its lessons, it is incomplete and often misleading. In a case of peritonitis, for example, the surgeon removes the offending appendix and drains the abscess cavity, but does he remember that such a suppurative process may produce myocardial, renal, and hepatic lesions which may chronically incapacitate the patient? The clinician trained in general pathology will see the cloudy swelling, fatty degeneration, and even necrosis which have taken place in organs distant from the site of trouble. This is undoubtedly one of the reasons for failure of the patients to return to complete health after cholecystectomy. The presence of adhesions to the stomach, transverse colon, etc., need not be especially discussed, but induration of the head of the pancreas is so important that a few words may be valuable. Briefly, there are two theories concerning its origin: The one, that infection reaches the pancreas from the common duct through the ducts of the pancreas; the other, that the infection is from direct lymphatic extension from the gall-bladder downward. If any one doubts that there is such a connection, they can assure themselves of it at any time, at least in the dog and the rabbit, by placing India-ink within the gall-bladder, tying off the cystic duct, and allowing the animal to live for three or four days. India-ink granules will then be found in the lymph channels extending down the gastrohepatic omentum and in the lymph-nodes down to and into the head of the pancreas. Dr. Deaver has spoken of the clinical pictures which he has traced in so many cases of cholecystititis: lymphangitis at the junction of cystic and hepatic ducts, lymphangitis and lymphadenitis along the gastrohepatic omentum, lymphangitis at the head of the pancreas, chronic pancreatitis. There is no doubt, of course, that the reflux of bile or other substances, including inflammatory products, into the pancreatic duct will produce pancreatitis; in fact, the injection of bile into the pancreatic duct is the classical way in which acute hemorrhagic pancreatitis or, as it is probably better called, acute hemorrhagic pancreatic necrosis can be produced, and no doubt a chronic pancreatitis will result by, let us say, submaximal doses, but that this is the usual mode of production of chronic pancreatitis is open to grave doubt. The anatomic make-up of the papilla of Vater and the relation of the opening of the pancreatic duct vary. Sometimes it empties into the common duct; sometimes into the papilla of Vater, but also occasionally in human beings, it enters the duodenum in such a way that reflux of bile from the common duct into the pancreatic duct is impossible. It would take

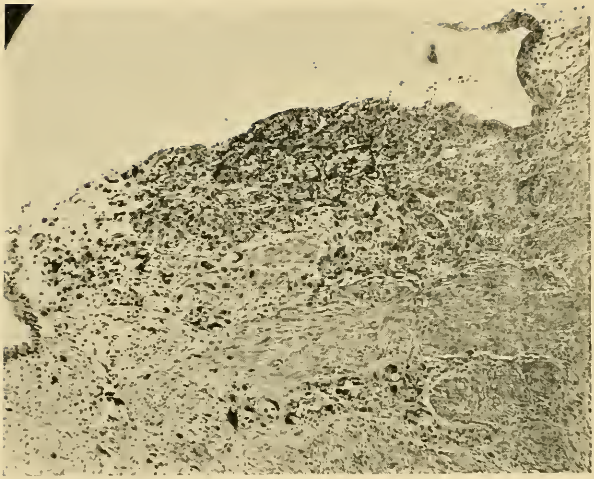

Fig. 21.—Primary carcinoma of the gall-bladder showing a small piece of normal mucosa at either end of the tumor mass which had infiltrated, as shown in other sections, through to the serosa.

but one case in which there is a chronic pancreatitis of the head, with a pancreatic duct so situated that bile cannot enter it, to prove absolutely that pancreatic infection can be due to lymphatic extension. We are on the hunt for such a case.

Finally, a few words may be said concerning primary carcinoma of the gall-bladder. We have not had the opportunity of examining any specimens from the bile-duct or the papilla of Vater because they could obviously not be removed. We have 9 specimens of carcinoma of the gall-bladder. Of these, 3 are of the variety known as adenocarcinoma, that is, they are composed of cylindric mucus-producing epithelial cells arranged in a more or less typical glandular form. Five are of the kind which we designate carcinoma simplex, that is, grossly, they were hard, almost cartilaginous, and histologically were composed of smaller, more or less undifferentiated spheroidal cells growing in groups and masses into a dense connective tissue with no attempt at differentiation into any typical gland-like structure. The ninth was of the type of squamous epithelium. This last tumor is quite interesting, but not excessively rare. It would lead us too far afield to discuss the theories of the transformation of cylindric to squamous epithelium, most of which theories have to do with the influence of chronic irritation. Gall-stones were present in this as well as in 6 other cases. The two gall-bladders which showed no stones were the seat of tumors of the adeno type. The remarks which were made under gastric ulcer and carcinoma will suffice for our ideas of the relation of chronic irritation and inflammation to the development of these neoplasms.

TRIALS, TRIBULATIONS, AND JOYS OF A SURGEON

In looking back upon his early career it is difficult for the surgeon to say whether his trials overshadow his triumphs or vice versa. It is but natural that, fired by youthful enthusiasm, the young man stands in danger of being more elated by his first real success than discouraged by his failures. With the passing of time, however, the tendency is to accept success with the humility born of the consciousness of one's limitations. Also if the triumphs are encouraging, the tribulations should be no less so, in that they should inspire one to greater effort and concentration. It is by our mistakes we learn.

From another point of view I may say that if thirty or even twenty years ago I had been asked whether the trials of the surgeon overshadow his joys, I might have been less keen about the joys than I am today. There is little doubt that the development of the science of surgery within the last several decades has added a degree of certainty of results to our operative technic scarcely dreamed of in surgical circles even as late as the days immediately following the epochal discovery of Lister's antiseptic treatment of wounds. This, of course, marks the definite transition of surgery from the realm of empiricism into the dignity of a science. It is by no means a reflection on the great men of pre-Listerian times to use the term "empiricism" with regard to their work. On the contrary, one cannot but admire their courage in the face of frequent failure and their steadfastness in the face of determined opposition.

It was no small thing, for example, for McDowell and later the Atlees and their contemporaries to continue to perform ovariotomy in spite of the abuse and criticism that surrounded them. Their success demands our utmost respect and admiration. Their determination paved the way for the more helpful and extensive pelvic surgery now at our command. Inspired by their pioneer work, gynecology was probably the first specialty to profit by the introduction of modern surgical technic, particularly as developed by that gigantic figure—Lawson Tait.

There is enough justification to speak of early surgery as empiric, for it is a well-known fact that although in the heyday of Greek civilization the surgeon and the physician were held in equal esteem, there came a long period in which this equality disappeared, to the disadvantage of the surgeon. He became the servant of the physician. It happened, however, that every now and then there arose a few men who by their social standing and superior knowledge and skill forged a place for themselves on a par with the best physicians of their time. Gradually the number of such men increased. Today there is no longer a question of equality between the status of the internist and the surgeon. Both are physicians conscious of the responsibilities which the practice of their art entails. Of both are demanded as necessary personal attributes to success, courtesy and gentleness, steadfastness and firmness in principle, cleanliness of thought and of living, generosity toward the poor, affability without servility to the rich, and a pleasing manner toward all fellow beings. The educational requirements today are also pretty much along the same lines, except for the superior technical skill and manual dexterity demanded of the surgeon *per se.* The wealth of sister sciences that have developed in our times undoubtedly has added to the triumphs of surgery, but there is danger of smug satisfaction at our progress. Oftentimes when I find myself becoming too well satisfied with things as they are, I am reminded of the declaration of a famous French surgeon made over a century ago that surgery had then almost if not actually reached the highest degree of perfection of which it was capable! This before the introduction of anesthesia! Before Pasteur and Lister! Certainly it contains a whole sermon against unhealthy contentment. At the same time there is no gainsaying the fact that there is a legitimate satisfaction in realizing that the surgeon of today is equipped with means of diagnosis and treatment which, even compared with the more immediate past, should make his yoke relatively easy and his burden relatively light. A picture of surgery as practised not only by the barber surgeons, but even in more recent times, represents such a story of horror and despair that it is remarkable not only that men should have been found willing to endure the tortures, but that there should have been those willing to attempt to operate, faced as they were with the greater assurance of defeat than of triumph. With the introduction of ether and of chloroform anesthesia it was only natural that the surgeon should seek to enlarge his field of operative work. But at every turn he found himself hampered by that grim specter, then known as hospital disease, but more familiar to us as wound infection. This, however, underwent a radical change when Lister, armed with Pasteur's divine rod, threw down the barriers in the path of surgical advance. And thus one of the greatest trials of the surgeon was translated into the opportunity for greater and greater triumphs. The entire method of surgical technic experienced a complete revolution, not in a day or a year, but through almost a decade of tribulation for the progressive man, confronted with the hostile criticism and reactionary complacency of his opponents who were as loath to adopt the new technic as they were to discard their blood-stained, infection-soaked operating coats.

But the new finally triumphed over the old, and Lister himself lived to see his method adopted throughout the civilized world. The modern surgeon, standing in his marble-walled, germ-proof operating theater, in his sterilized gown, mask, and gloves, and sterile armamentarium, is like a general armed *cap a pie* for the battle against insidious disease. Surrounded by an efficient and intelligent corps of uniformed assistants, nurses, and orderlies, all of them similarly arrayed in sterilized washable gowns, his is but to command and his orders are obeyed with military precision. But, like a general, he himself must bring to the operating-table a detailed knowledge of every strategic point not only within the limits of the immediate field of battle but of the surrounding area as well. In surgical language this means primarily an exact knowledge of anatomy and anatomic relations, coupled with no less exact a knowledge of physiology and the physiologic effect of the procedure which he proposes to carry out. Nature, like all females, is very sensitive. While she very often will tolerate man's interference to a surprising degree, there are serious limitations to her tolerance, and then she rebels. It is the duty of the surgeon to know these pitfalls; only by avoiding them can he hope to win the battle.

If the surgeon has risen to the plane of social and professional equality with the so-called internists, it is because he has ceased to be a mere mechanician. His ever-increasing experience in comparing living pathology as seen at the operating-table with clinical signs and symptoms, offers the opportunity of greater certainty in diagnosis; this likewise gives him greater acumen in making a strictly medical diagnosis. Indeed, it is as essential for success that the surgeon be well-grounded in medical diagnosis as it is for the internist to be versed in surgical diagnosis. I have time and again made and shall continue to make the plea that the internist take every opportunity to accompany his patients to the operating-table. Thus only can he acquire skill in recognizing surgical conditions, and thus will he be able to see with his own eyes the evil effects of procrastination. For it is the many cases that are brought too late to the surgeon that turn what might be his joys into tribulations.

It may be true as is often asserted that the surgeon like the poet is born such. But, on the other hand, it is for the surgeon to choose to surround himself with men who are imbued with the real love of the profession, true physicians, willing to spend years in apprenticeship, and to be receptive to orders from a superior; men who realize that only after arduous, prolonged systematic training is it possible to become a leading factor in a profession which has reached its present position only after centuries of endeavor. It may well be counted among the joys of the surgeon to train worthy successors in the work he loves so well. Personally, some of my pleasantest and most satisfying recollections are the hours spent in clinical work among my students. It has been my endeavor to imbue these young men with the sacredness of their calling and to impress them with the fact that it depends upon them to develop the surgery of the future. If I have succeeded in inspiring only a few with the real spirit of devotion and dedication to scientific truths, and if only one or happily two, become real leaders, I shall deem that I have contributed something worth while to the honor of our calling and to the surgery of the days that are to come. This training, however, does not stop in the class room; it must be carried on throughout active life. In his service at the hospital the surgeon not only must surround himself with capable assistants, but it is most important that by means of conferences with his staff he carry on a periodic survey of the activities of his department in order to get a proper estimate of results, be they successes or failures, of his work. This interchange of opinion, deliberate discussion, and introspection are of the utmost value in stimulating the staff to greater and greater efficiency. Success means greater effort to continue the good record; failure means still greater effort to prevent its recurrence. The problems presented by this review open the way for experimental research by which the why and wherefore of results of certain line of treatment can be ascertained: We can only hope to profit by our mistakes by developing methodic habits of thinking and of working. It is in this way that medicine and surgery may become more and more exact sciences.

It is to the credit of the American College of Surgeons that this line of endeavor has become a part of their hospital standardization program. It is gratifying to note also that this special tendency is receiving recognition among medical and surgical societies abroad. Sir Harold Stiles, at the Convocation of the American

College of Surgeons held in Philadelphia in October, 1921, speaking of the need of Co-operative Thought in Surgery, says: "The possibilities which your College affords for organization and prosecuting a systematic investigation not only into immediate and end-results of operation but also into the value of various other measures employed in surgical therapeutics are very great. . . . With the machinery for organization which your College possesses, the untold wealth of material at its disposal, and the reliable observations which could be guaranteed, I am convinced that . . . America would again lead the world. . . . Science is ever becoming more and more international. America has taken more advantage of this cosmopolitan spirit than we Britishers, and in my judgment this is the main reason why the scepter of surgery is today wielded by America." This is flattering, encouraging, and stimulating. The stimulation is needed, and where it is most needed is in relation to better and more complete histories than are found in the record rooms of our hospitals. It is the paucity of information contained in the average history that seriously interferes with the success of the conference plan just alluded to. It also will interfere with the realization of the flattering prediction of Sir Harold Stiles that America is to wield the scepter of science and surgery of the future. It is only by carefully taken histories combined with systematic follow-up records that valuable data in large numbers can be collected to serve as the basis of scientific investigation in the advance of medical knowledge. This subject of history-taking ever has been, and probably always will be, a source of tribulation. Sometimes it seems to me that the defect complained of can be remedied by the use of standard history forms for various conditions, on which all the data that may be desired are printed so that the completeness of the record does not depend upon any one individual. While this method might do away with careless and inaccurate history-taking, it would, however, have the defects of its virtue in that it would fail to train the intern (when history-taking is dependent on the intern) in the habit of expressing in his own language the information gained from the patient—a habit that should be stimulated and fostered. Personal responsiblitiy is so essential a part of the surgeon's life.

Indeed, every surgeon must accept the responsibilities which his profession entails. This responsibility, to my mind, is even greater than that of the internist. It cannot be gainsaid that in no other profession and in no other branch of medicine does the individual so confidently place himself at the mercy of his fellow-man as when he places himself in the hands of the surgeon. With this control of human life in his hands the burden of the surgeon is truly a heavy one. For this reason if for no other is it essential that except in emergency cases the surgeon should apply all his accumulated knowledge to the study of his patients before operation; and that the preparation for operation be as faultless as it can be. It is for this reason that surgical diagnosis assumes such immense proportions in our work. Not until the diagnosis has been made with a fair degree of certainty should operative interference be considered, and when considered it should be done at a time only when the risk entailed gives reasonable assurance of future well-being. At this point I would again emphasize the importance of the practitioner's being able, if not to make a surgical diagnosis, at least to recognize when a condition becomes surgical. The tribulations of the surgeon would certainly be materially reduced if his professional brethern would realize the importance of referring their cases early, instead of sending them after prolonged medical treatment has proved futile. Too often is the surgeon called upon to use the knife as a last resort, to be confronted with failure. How often have I been called upon to operate upon a perforated appendix which, starting as a simple acute inflammation a few days earlier, was treated or, rather, maltreated by purgation until the poor appendix burst with angry resentment. Instead of the simple removal of the appendix then we have all the dangers of peritonitis, infection, and the like to deal with; and instead of the justified expectancy of complete recovery from a simple operation, we have to fear operative results such as fecal fistula, intestinal obstruction, or the more remote one of hernia—surgical trials which might well have been avoided. Or, again, we are called upon to perform an emergency operation in a case of perforating duodenal or gastric ulcer. Subsequent inquiry reveals the fact that the patient in question had been presenting ulcer symptoms for years, and instead of having been given the benefit of early surgery the disease has been allowed to progress until perforation or hemorrhage with its attendant dangers had set in. How often also has malignant disease been allowed to progress unhindered while it was being treated for syphilis? The risk to life from early operation is assuredly less than the risk to economic and social usefulness of the same operation postponed.

Let us then suppose that our internists send their patients early and let us suppose that the history has been well taken, and the details have been properly correlated with the results of clinical and laboratory studies. The next concern or trial of the surgeon is the operating-room. I wish here to correct an erroneous impression that may have been obtained from my previous utterances, that I do not place much reliance on laboratory findings. Although I do not hesitate to point out the shortcomings of laboratory tests and insist that they are only of relative importance, I certainly do appreciate their inestimable value. Without the laboratory in all its phases medicine could not possibly have made the strides it has. Moreover, I believe that teamwork between the internist, the laboratory man, and the surgeon is not only essential for the success of all three, but likewise serves the best interest of the patient. Ever mindful of our responsibility and with our desire to give the patient the best that surgery can offer, it is imperative that the operating-room be under the supreme control of the surgeon, assisted by a competent staff and by well-trained nurses. A minor slip in technic may mean at the least a wound infected; and the most, a life lost. It is the realization of this fact that imparts a sort of reverence to a well-conducted operating-room. To me it is as sacred if not more so than the Church. The Ritual of An Operation is the significant title of a significant essay by Sir Berkeley Moynihan on this subject. He describes the surgeon as a "ritualist," who, while properly loyal to the ordinances of his surgical sect, must not be unduly concerned with the prescribed forms and ceremonies, but continually seek for simpler methods and a better way. "In the craft of surgery the masterword is simplicity."

Simplicity in surgery is best obtained by well-studied, careful, and clean technic. For in this way can we best insure prompt healing, undisturbed convalescence, and the consequent pleasanter memories of the entire ceremony. It would be superfluous at this date to emphasize the necessity of rigid enforcement of the wearing of gowns and face-mask, not only for the personel of the operating-room, but for visitors as well; or as Sir Berkeley expresses it, the high-priest, the acolytes, and the congregation. Among the assistants or acolytes we must include (if we wish to continue the metaphor) the white-robed vested choir—the nursing staff. Lack of attention on the part of the latter is like a false or discordant note in a hymn, apt to throw the entire ceremony out of harmony. Next to the surgeon himself the most important person in carrying out the operation is the anesthetist. The nurse anesthestist has become an established feature of nearly all surgical services in this country. In my experience it has proved perfectly satisfactory. While her responsibility is great, it should never include the choice of the anesthetic to be used. This must devolve upon the surgeon who is best acquainted with the patient's requirements. I have become a firm believer in selective anesthesia. If anoci-association will save a particular patient anxiety or shock, that patient should have it. If nitrous oxid gas will lessen the risk for another, the surgeon must submit to the less favorable conditions of operating and must order gas anesthesia. The responsibility of an anesthetic death rests as much on the surgeon as on the anesthetist, and perhaps more so now that the nurse anesthetist is almost universally employed instead of the trained physician, as was formerly done. It is not the fatalities alone, however, which the surgeon must consider, for, on the whole, these are rare, but the state of the patient; the anesthetic, therefore, must be chosen for each individual case.

A source of trial to the surgeon often is that the young assistant of today is apt to concentrate all his attention upon the operation itself to the exclusion of what may come after. He fails to realize that the operation is but the first stage in the battle. Of course "well begun is half done" applies here as elsewhere, but no matter how brilliantly and dexterously an operation may have been performed the postoperative course is of no less importance, particularly to the patient, who in the last analysis is the chief person concerned in the ceremony. For what profiteth it the patient if the operation is a success and the postoperative treatment disastrous? Nor do our responsibilities cease when the patient is discharged from the hospital a happier and more hopeful being than when he entered it. We still have a duty to him, to ourselves, and to those who will come after him and after us. The immediate duty to the patient and to ourselves is to give careful instructions as to posthospital régime, as to diet, mode of life, etc. This at the same time is our duty to ourselves, so that the result of the operation may be the best that can be expected. The tribulations of the surgeon would be materially reduced if this were routinely done and conscientiously carried out by the patient. For we would then hear much less of postoperative return of symptoms. It is only natural that a patient who had been suffering for years from ulcer or gall-bladder symptoms should be warned of the necessity of care in his diet for some time after operation. The disabilities of months and years of suffering cannot be suddenly dispelled by the operation; and the minimum disability of the operation itself must also be considered. Our remote duty to the patient and to those that come after him implies keeping in touch with him by means of a systematic follow-up system. The results of treatment will be reflected in its remote results, and will be useful in the selection of operation and other treatment for future patients. The joys of the surgeon are materially enhanced by seeing with his own eyes what his skill has been able to do in restoring a pain-racked debilitated body to health, vigor, and usefulness. As I have already intimated, the remote results will materially affect surgical advance of the future. Statistics in the mind are not the same as on paper, in spite of the fact that it is often said that we can prove whatever we wish by statistics. But an honest survey and record of remote results as found in the follow-up history are indisputable evidence of the work of our hands. The emphasis in postoperative results has shifted from mortality to morbidity, and we no longer can consider an operation successful which leaves the patient a chronic invalid and incapacitated. If our follow-up results in operations for chronic appendicitis, for example, show that a certain percentage of patients, after a reasonable time, have not been relieved of their preoperative troubles, we cannot help knowing that our treatment has not been the best for that type of patient, even though the operative mortality in the series had been *nil*. We realize that we must modify our treatment the next time we meet with the same governing conditions, such as symptoms, age, occupation, etc., of the patient.

Thus the trials and joys of the surgeon are not only those within the hospital, but extend in time and place beyond it. Nor are they all concerned only with his actual operative activity. There are other sources of tribulation over which he has little if any control. I refer to the numerous stumbling-blocks in the path of surgical progress due to lay ignorance and fanaticism, as evidenced chiefly in the agitation against animal experimentation. The result of this anatagonism on the part of misdirected individuals is that large fields for investigation which are essential to the advance of surgery have been untouched. It would be useless for me to enumerate the many contributions to present-day methods that have come from animal experimentation. It is only by unhampered zeal in future experimental research that we can hope to carry on the lighted torch which has been shining so brilliantly in the past. "A duty," says the poet, "is a demand of the hour."

It is the duty of every physician to see that any effort, whether lay or otherwise, which has for its function the limitation of medical research should be checked. Huxley, in discussing the opposition of the antivivisectionists to the proposal to contribute to the support of the Pasteur Institute, said, "It proceeds partly from the fanatics of the *laissez faire* type who think it better to rot and die than to be kept whole and lively by state interference; partly from the blind opponents of properly conducted physiologic experimentation, who prefer that men should suffer rather than rabbits or dogs; and partly from those who for other but not less powerful motives hate everything which contributes to prove the value of strictly scientific methods of inquiry in all those questions which affect the welfare of society." I heartily subscribe to this statement.

If the trials of today are to be made the joys of tomorrow, clinical and experimental research must go hand in hand. For how else could the direful preantiseptic days of Lister have been turned into the joyous days of our present aseptic method? Compare our knowledge of the diseases of the abdominal viscera of a quarter of a century ago with that of today, and the truth of these statements becomes apparent.

With trials and responsibilities come pleasures and compensations. It is a joy to know that we are members of a profession which demands untiring patience, constant self-denial, and unselfish industry. And if we must be warned of our stumbling-blocks, we can be cheered by our advances. A long and weary road must be traveled, but many of us find happiness in the end.

It is a joy to know that even though we may still be frequently regarded by our friends, the general practitioners, as no better than the barber surgeons of yore, we daily have it in our power to be the means of correcting mistakes in the interpretation of the language of living pathology, and thus save an otherwise condemned sufferer from medical procrastination. We can point with pride to the contributions to our knowledge of the physiology and pathology of the abdomen, for example, which have come from the operating-room. It is only by standing at the elbow of the surgeon that the internist can share with the surgeon the information thus obtained, and gain proficiency in diagnosis and the proper conception of treatment. As I have said before, cooperation between the surgeon and internist and the laboratory should go hand in hand if we are to advance and improve still further on our present-day procedures of which we are justly proud. Our greatest joys will be obtained when the intimacy and intercourse between these is increased.

What a pleasure it is to know that surgery is daily adding to the sum total of human knowledge, to know that "pain, hemorrhage, and infection, the three great evils which had always embittered the practice of surgery and checked its progress," are now robbed of most of their terrors.

Our calling demands the high intellectual attainments and noble character which is demanded of all those who choose to work for the benefit of mankind. The great deeds of surgery have been the result of zealous enthusiasm, and the contributions of surgery equal those of any of the other sciences and arts. While we may not all attain the same eminence in the profession, the honors are shared by all, for we are all part of a great enterprise, as Ambroise Paré has said, "beautiful and best of all things is to work for the relief and cure of suffering." There is ample work to do and ample joy for all the tribulations we encounter, for—

"Little do ye know your own blessedness, for to travel hopefully is a better thing than to arrive, and the true success is to labor."

SOME SURGICAL CONDITIONS OF THE INTESTINAL TRACT

The subject of my paper today is such a broad one that I feel privileged to select those conditions for discussion which are of particular interest to me, and to omit others which are of no less importance, but which time does not permit me to discuss. I will therefore group my remarks under three headings: (1) Inflammatory lesions of the intestinal tract, (2) malignancy, and (3) tuberculosis, which for the sake of clearness I have excluded from the inflammatory lesions.

One of my main reasons for choosing these particular aspects is that I have felt and feel with an increasingly strong conviction that there are certain lesions of the intestinal tract which, if diagnosed early and operated upon promptly by a competent surgeon, would no longer be the chief causes of our surgical mortalities. The success of modern times in checking the ravages of the white plague must be attributed to the educational campaign carried on among the medical profession as well as the laity, and this success should be duplicated and even surpassed by a similar campaign conducted particularly among the profession, covering the subject of early diagnosis and prompt intervention in surgical conditions of the intestinal tract. The effect of delay in such cases cannot be better illustrated than by a discussion of appendicitis. It would also be quite unnatural and quite contrary to the habits of my surgical life were I to miss an opportunity to have a dig, if only in a figurative sense, at that inoffensive looking organ which is so deceptive and so potentially harmful, and which, excepting perhaps the tonsils, has become the most frequent sacrificial offering of the human body to the god of prophylaxis. But when this sacrificial rite has been omitted and the appendix becomes the starting-point, as it is so often does, of bacterial invasion, we may expect to find the typical picture of inflammatory disease of the intestinal tract and the peritoneum with all its dire and dreadful possibilities.

It is now thirty-four years since Reginald Heber Fitz, of Boston, first demonstrated that the most frequent cause of pain in the right iliac fossa is appendicitis; he also laid down the principle that in the presence of peritoneal involvement it is best to wait to operate until localization of the process. It is a stigma to American surgery that John B. Murphy in June, 1915 reported the average hospital mortality for appendicitis as "just a little over 10 per cent.," and I must remind you that this included appendicitis of all types that came to the hospital for operative treatment.

The appendix cannot be said to have any definite position. In the rotation of the cecum from the left to the right side and its descent into the right iliac fossa, the appendix may be arrested in any one of a number of positions. My early years were spent in the anatomic laboratory, and in my work there upon the cadaver, as in my subsequent work with the living subject, I find that the appendix usually occupies one of five positions: (1) The most common and probably the normal position is to the inner side of the cecum with its tip pointing toward the spleen, and behind the termination of the ileum; (2) to the inner side of the cecum with the tip hanging over the brim of the pelvis; (3) to the inner side of the cecum with the tip near Poupart's ligament or curled up to the inner side of the cecum; (4) lying along the outer side of the ascending colon; (5) lying behind the cecum and the ascending colon in the retrocecal and retrocolic fossæ.

The exact point of attachment of the appendix to the cecum depends upon the type of the latter, since the infantile cecum is conical and the appendix extends off from its apex. Usually the outer side of the cecum grows more rapidly than the inner side, which accounts for the more usual attachment of the appendix to its inner side.

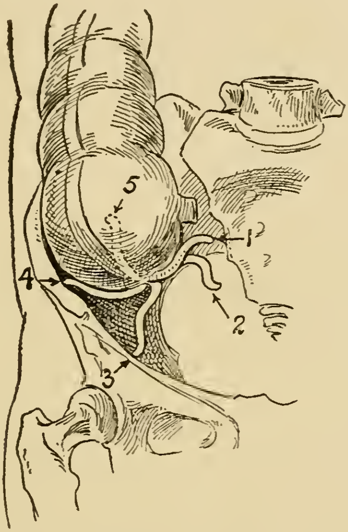

Fig. 22.—Illustrating the five positions of the appendix.

These positions have more than a theoretic interest. They are of importance not only at operation, but they have a marked influence on the symptomatology. The pain from an inflamed viscus without irritation of the parietal peritoneum is referred through the sympathetic nerves to the spinal cord, and then referred to the peripheral distribution of the spinal nerves arising from those segments of the cord with which that viscus is connected through its sympathetic supply. The initial pain in appendicitis, for example, is always referred to the umbilical area. However, when peritoneal irritation subsequently occurs, the pain is localized over the position of the appendix and the area of involved parietal peritoneum. Therefore, while the pain in the majority of cases of appendicitis localizes in the right iliac fossa, it sometimes localizes high up, simulating disease of the gall-bladder, or low down, as in pelvic disorders.

The tenderness elicited by deep pressure over McBurney's point arises from pressure stimulation of the nerve-endings in the inflamed parietal peritoneum. This tenderness is an indication of the actual position of the appendix, and therefore by it we are able to locate more accurately the position of the organ. Burgess compares the rigidity of the abdominal wall in appendicitis to "the muscular spasm around a joint in a state of acute synovitis. This is nature's effort at protection, fulfilling the same purpose—in fact, the peritoneum is the great visceral joint."

Early vomiting, like the early pain, is also a reflex phenomenon. A recurrence of vomiting later in the attack is a grave symptom, indicating the onset of a spread of the inflammation to the peritoneum.

Among internists, and also among surgeons, there is a tendency to assign a relationship between the leukocyte count and the extent of the lesion. Personally, I am guided rather by the physical sign of exquisite local tenderness than by the blood-count, except that a low count in the presence of grave symptoms and with evidence of peritonitis is a danger signal, indicating lowered resistance, and usually making operation unsafe. While, generally speaking, a leukocytosis of more than 15,000 may indicate acute changes or a rapidly developing peritonitis, it does not indicate the extent of the inflammatory process nor the imminence of perforation or of gangrene. Unless recognized in time and subjected to prompt surgical treatment the inherent danger of appendicitis is extension into and involvement of the peritoneal cavity. It is, of course, the danger of peritonitis that gives to appendicitis its grave importance.

Although we now know that the peritoneum possesses remarkable powers of resistance because of the bactericidal action of its serum, its resorptive power, and its tendency, aided by the omentum, to form protective adhesions, there are cases, especially those with a perforation at the base of the appendix, in which the infection is overwhelming. Also sometimes the infection when streptococcic, for example, is so virulent that even without perforation the peritoneum is so flooded with extending infection that no encapsulation occurs and a diffuse peritonitis results. These are the end-results of procrastination in the treatment of this disease. The majority of the cases of appendicitis that die in our hospitals do so because they have come too late for a successful operation. The early symptoms are usually unmistakable. If, when they appear, the patient were subjected to immediate operation, our mortality statistics would doubtless be much improved.

One of the factors that favors the development of pus, perforation, gangrene, and peritonitis is the practice of purging the "acute abdomen." Purging a patient who has symptoms of appendicitis is little short of criminal. The campaign against it, I am glad to say, is beginning to show results. Nevertheless at the risk of tiresome repetition I do not hesitate again to emphasize this point. Every effort of nature to protect a diseased abdominal viscus by rest is frustrated by the peristalsis excited by the action of strong cathartics and purges. Purgation and perforation are, in my experience, almost synonymous:

> "Purgation means perforation
>   In an appendix kinked and bad.
> Food and drink worry him
>   And aperients drive him mad."

The ideal treatment for all lesions which threaten the peritoneum is to operate before the infection has established an independent footing in the peritoneal cavity. After the early and most favorable moment has passed the best time for operation will depend on the extent of the peritoneal process in each individual case. Appendicular inflammations have a strong natural tendency to localize, so that rather than operate in the presence of an acute diffusing peritonitis (where there are as yet no distinct signs of localization, or of locating the lesion) with the risk of spreading the infection to areas as yet uninvolved, it is preferable to await further or definite localization. In localized peritonitis, whether free or confined, operation may be undertaken at once. I need scarcely say that for generalized peritonitis there is no operative treatment, nor can incision and drainage limit the advance of the acute spreading type of the disorder. By operating we may remove the appendix, which, no longer dangerous, is a secondary consideration, but the trauma inflicted and the depression of vitality are such as to lose the battle between the advancing inflammation and the patient's power of resistance. With rest the diffuse inflammation subsides and becomes centered about the appendix. This can best be accomplished by the usual routine of complete physiologic and anatomic rest, supplying fluids to the body by means of proctoclysis and hypodermoclysis intermittent or continuous, and the administration of minimal doses of morphin to assuage the pain and inhibit peristalsis. Those of you who have not used it have much to learn from the combined Murphy-Ochsner-Fowler-Alonzo-Clark-Crile method of treatment of peritonitis.

As to the operation itself, neither the method of approach nor the question of drainage can be formulated by rule. The incision will depend upon the degree of infection. Early cases which show a limited amount of turbid fluid without any odor do not require drainage. Actual pus should, of course, be drained. For pelvic drainage I use a glass tube, elsewhere rubber tubes. But the entire question of drainage is one for each surgeon to decide for himself in a given case, guided by experience and judgment. I might say in passing that where there is a localized abscess in the right iliac fossa, I prefer the extraperitoneal approach, making an oblique incision close to the anterior superior spine, disregarding the muscle-fibers, and opening the abscess cavity well to the outer side. In this way the general peritoneal cavity is not contaminated nor is the ileum brought into the wound. It is true that some of these patients develop a postoperative hernia, but it is better to have a live patient with a hernia than a dead patient without one.

Postoperative treatment can best be summed up in the words "Let the patient get well himself." The essentials to this rule are the semi-sitting position, enteroclysis, hypodermoclysis, nothing by mouth until peristalsis has been restored morphin in minimal doses, careful nursing, and watching for complications. As soon as peristalsis sets in, as evidenced by auscultation and the passage of flatus, fluids may be given and enteroclysis discontinued. I do not believe in the early removal of drainage, and I always tell my house doctors that it is better to let the drainage rot out than to remove it too soon. I want to caution you against giving enemas too early after operation in bad cases. I have seen an asafetida enema emptied into the peritoneal cavity because of its too early administration.

Peritonitis, of course, may result from inflammation of any portion of the gastro-intestinal tract. Very occasionally it originates from ulceration of the small intestine or of the large bowel. I have already discussed the perforation of gastric and duodenal ulcers in a previous lecture, but, excluding appendicitis, perforation of peptic ulcer is the most frequent cause of peritonitis. However, there are other less common ulcerations and inflammations which I wish to mention.

Primary ulceration of the jejunum is exceptionally rare, in fact, I have never seen one, but jejunal ulcer following gastro-enterostomy for peptic ulcer is more frequent. In the first type the history rarely contains any indication of the presence of the ulcer until perforation occurs. In the second type the ulcer may develop early and perforate shortly after its development. The perforation of a typhoid ulcer is also a cause of peritonitis. The most frequent site of this perforation is in the ileum, within 12 inches of the ileocecal valve. Although perforation of a tuberculous ulcer is only occasionally seen, it occurs in from 5 to 10 per cent. of the cases of intestinal tuberculosis.

Somewhat less rare but by no means frequent is ulceration of the large bowel, the favorite sites of which are the cecum and the sigmoid flexure. German writers believe that there is a tuberculous element in these, and, in fact, I have seen a patient develop tuberculous peritonitis after recovery from an ulceration of the cecum, the pathologic laboratory report having stated that no evidence of tuberculosis was found in the ulcerated area.

In view of the fact that Meckel's diverticulum persists in from 2 to 3 per cent. of all individuals, it should be mentioned as one of the possible intestinal sites of inflammatory disease. Diverticulitis of the large bowel, especially of the sigmoid, has been encountered by every surgeon of large experience. Diverticula of the colon are a fairly frequent site of ulceration and subsequent perforation, causing a localized or generalized peritonitis. These lesions in the sigmoid are frequently mistaken for malignancy, and especially when, due to hyperplasia of the bowel, they cause an intestinal obstruction.

In arranging the topics for this lecture I have purposely omitted a general consideration of intestinal obstruction. I have chosen carcinoma and not sarcoma as an example of malignancy of the intestinal tract because it is so much more frequent. In 26,340 instances of carcinoma collected by Stengel, 2225, or 8.56 per cent., were intestinal. While any portion of the intestine may become the site of this tumor, the cecum, the appendix, the sigmoid proper, the rectosigmoid, and the anal region are the most favored places. Only about 4 per cent. of all intestinal carcinomata occur in the duodenum, while 3 per cent. occur in the jejunum and the ileum. Like malignant disease elsewhere in the body, carcinoma of the bowel occurs chiefly after middle age, though it is not infrequent in early life. The age incidence of rectal carcinoma, however, is usually somewhat later than that of carcinoma of the colon. A definite tissue predisposition appears only in those cases having intestinal polyposis, and, as Kraske has pointed out, there is no satisfactory evidence that cancer develops in tissue altered by hemorrhoids, fistulæ, or cicatrices. He believes that the early symptoms commonly attributed to hemorrhoids are due to a slow-growing rectal carcinoma. Personally I am not sure that this is so, but, on the other hand, it is a rare thing to have a patient suffering from an intestinal carcinoma give a history of a previous ulcerative proctitis or long-standing colitis.

At the ileocecal valve, the junction of the appendix, the rectal folds, and the anus there are more or less definite interruptions of the structure of the intestinal wall, and the anatomic arrangements may influence the development of tumors in those regions. The greater predominance of carcinoma in the large bowel compared with the small bowel is also undoubtedly related to the anatomic curves, the normal delay in the passage of contents, and the numerous organisms present, all of which predispose to irritation, the fundamental cause of malignancy.

Keith has called attention to three points in the large bowel where normally the contents are delayed: (1) near the commencement of the transverse colon, leading to filling of the cecum and the ascending colon; (2) at the rectocolic junction, leading to accumulation in the pelvic colon; (3) above the anal canal, leading to accumulation in the rectum. These points of normal delay correspond to the more frequent sites at which malignant disease is found to occur. I think it very likely that the chief factor in the production of carcinoma at sites such as the cecum and the hepatic, splenic, and sigmoid flexures, and the rectal valves is irritation acting at relatively fixed points in which delay or stasis occurs, together with a higher specific gravity of the fecal current in the large bowel.

It is the insidious growth of intestinal neoplasms that makes them so dangerous. Frequently the patient has been treated over long periods for a supposed gastric condition. Indeed, perforation or obstruction may be the first actual sign which induces the patient to seek medical advice. Erdman and Carter, in a recent report of 129 cases of carcinoma of the colon, observed 3 instances (21 per cent.) of perforation, all of them occurring from a growth in the sigmoid. Permit me to recite a recent personal observation:

Case No. 816/22. Female, aged thirty-five years, admitted to the Lankenau Hospital March, 1922, with the following history: One week before admission the patient was seized with chills, fever, pain in the back and across the lower abdomen, which finally localized in the lower left abdomen. Accompanying symptoms: burning and cutting sensation on urinating, slight urinary frequency; bowels habitually constipated. Menstrual history: Periods regular, not painful, no leukorrhea. The patient has had 2 children and one miscarriage. Her past medical history was negative except for appendectomy four years ago. Physical examination negative except the abdomen. This was distended, and a soft, slightly movable mass was palpable in and to the left of the pelvis. Vaginal: A fluctuating, slightly tender mass was palpable posterior and to the left of the uterus and a rounded mass could be felt protruding into the rectum. A diagnosis of tubo-ovarian disease was made.

An operation (March 15, 1922), through a lower right rectus incision, the mass to the left of the uterus was found to be an abscess with the sigmoid resting upon it. The cavity was evacuated, and a perforation was found in the sigmoid in communication with the abscess cavity. The sigmoid was brought up and hooked into the wound preparatory to a classic Mikulicz operation. The second stage was done on March 22, 1922 under novocain anesthesia. The protruding loop of bowel was excised with the cautery, an enterostomy tube inserted, and the opening closed. The final stage was performed on May 19, 1922, when an incision was made around the colostomy opening, the bowel was dissected free from scar tissue, and the proximal and distal ends united over a rectal tube inserted through the rectum. This time the operation was begun under ether anesthesia and finished under nitrous-oxid anesthesia. A cyst was also removed from the right ovary. The patient was discharged June 9, 1922, with a granulating wound, but without any fecal discharge.

The insidiousness of these growths can best be illustrated by the following circumstance: Four years ago one of our distinguished Philadelphia internists came to my office with a fellow-practioner who had merely stopped in Philadelphia in order to be "gone over" after a very difficult year's work. In giving his history he mentioned the fact that he suffered from constipation.

This led me to make a digital rectal examination, which disclosed an annular carcinoma, the existence of which was not in the least suspected.

Nearly all of these malignant neoplasms are secondarily infected. There may be a local or a spreading suppuration, a localized peritonitis, or a perforation into other viscera, such as the stomach, the bladder, or loops of the intestine. All these factors increase the operative mortality rate. They also are largely responsible for the cachexia which develops, since metastatic invasion is slow and late, and a patient may die with little evidence of high glandular or hepatic involvement. Thus, early recognition will make these tumors a fruitful field for the aggressive surgeon.

When we stop to consider the symptomatology of malignancy of the large bowel it would seem as though we should discuss each regional type separately. Clinically, however, this is unnecessary. There are some special features to which I wish to direct your attention. Carcinoma of the cecum, owing to the fluid state of the contents of the small bowel, rarely causes acute ileus. It is only when the ileocecal valve is affected that signs of acute obstruction are present. The cecal type of growth is occasionally mistaken for chronic appendicitis. This type is associated with an early marked anemia. The tumor is palpable only late, and the Roentgen demonstration is frequently of little value early in the disease.

While radiography is not infallible in the diagnosis of carcinoma of the colon, when positive it certainly offers valuable evidence of the presence of a filling defect in the affected portion of the gut. In some tumors at the hepatic and splenic flexures the overlapping of the barium shadows may prevent a satisfactory study. But I do not believe in giving a bismuth or barium meal for the purpose of radiographic study in cases where the tumor can be felt and the diagnosis leads to a decision to operate. The barium collects behind the obstructing mass and may offer serious difficulties during the operation.

The exact symptoms of intestinal malignancy are dependent upon the site and the progress of the neoplasm. The pain in the distal colonic growths may be abdominal, sciatic, lumbar, or perineal. Slight cramp-like pain in the lower abdomen occurring constantly or frequently should call for a rectal examination. Tenesmus and an unfinished sense after defecation is frequently an early symptom of sigmoid or rectal tumors. A symptom which is fairly constant in a carefully taken history is constipation alternating with diarrhea, the latter especially in the morning. Blood and mucus in the stool are often the first symptoms of the disorder, but owing to the fact that bleeding is a common sign of hemorrhoids, and hemorrhoids often accompany the tumor, the average physician is apt to neglect making a digital examination. Loss of weight and a constantly increasing low-grade toxemia due to stasis result in a cachexia the severity of which depends upon the site, the type, and the duration of the cancer.

Carcinoma of the rectum of itself rarely produces obstruction, although obstruction may be the first symptom of a tumor in the sigmoid. Occasionally ulceration may begin early and the sloughing of the growth may be sufficient to keep the lumen open. If obstruction is present in the lower rectum or in the anus, the feces are apt to be ribbon-like or shaped like small balls.

The value of a digital examination for a low tumor or a proctoscopic examination for one higher up cannot be overestimated. A rectal carcinoma is rarely situated entirely on the anterior wall, it usually also infiltrates the sides. A painful crater-like depression of a soft fragile tumor, or a hard irregular mass, which when touched causes exaggerated tenesmus, need scarcely be differentiated any further from a simple stricture.

Any operation undertaken for the cure of cancer of the bowel must be based upon the pathologic findings and should include all tissues that may have been invaded. It must be remembered, however, that operation, no matter how extensive, may not establish a cure. The cancer may spread in one of three ways: (1) Locally by direct extension through continuity or contiguity; (2) through the venous system; (3) through the lymphatic system. Although in young individuals carcinoma of the intestines, especially of the rectum, progresses very rapidly, in older persons it is comparatively local for a long period. The growth may exist for a considerable time without being discovered, and even when discovered the surrounding tissues are not necessarily involved. Extension through the venous system may occur at an early stage and result in metastatic growths, especially in the liver. However, this type is not frequent, and liver metastasis is generally a late manifestation. But the most important route of extension is through the lymphatics, either downward, laterally, or upward. Regardless of its position, cancer of the rectosigmoid and the rectum is apt to spread in all three directions.

The treatment of cancer of the intestine if seen early enough is essentially surgical. Tumors proximal to the sigmoid may be treated by a one- or a two-stage operation, depending upon the time the case comes to operation, that is, whether or not an acute obstruction is present. For growths of the sigmoid and parts distal to it I prefer the two-stage operation. There is no doubt in my mind that the patient's condition is improved by a preliminary colostomy. For the sigmoid, the rectosigmoid, and the rectal carcinomas four methods are available: (1) The abdominal route for the sigmoid tumors; (2) abdominoperineal extirpation is favorable in rectosigmoid growth cases; (3) in less favorable cases the perineosacral extirpation, and (4) in inoperable cases radium treatment, which, of course, is merely palliative. A preliminary colostomy should be performed regardless of which method is decided upon, except perhaps the first. The toxemia improves, the patient usually gains in weight, the inflammation is reduced. The pain and discharge are lessened, there is no distressing diarrhea, sleep and rest are obtained, improving the patient's general health so that not rarely inoperable cases may become operable. I believe in opening the abdomen. In no other way can we work under the guidance of the eye and accurately estimate the degree of dissemination. The question of end-to-end as against side-to-side anastomosis is a matter of individual preference. Personally I prefer the side-to-side anastomosis and would advise it whenever it is feasible.

Cancer of the rectum offers a better prognosis than does cancer at other sites of the intestinal tract, but in all cancers of the large bowel early diagnosis is essential. The failure of the general practitioner to act early is due either to carelessness or to ignorance, or to both. A lack of knowledge of the early symptoms and hesitancy to make a digital examination are often the cause of catastrophies.

The dictum that there are no hard-and-fast rules in surgery applies with particular force to the type of operation in the treatment of cancer of the rectum. Sir Berkeley Moynihan on being asked on one occasion what operation he used for excision of the rectum characteristically replied that he changed his mind more frequently with regard to the treatment of carcinoma of the rectum than with respect to any other matter in surgery. By changing the term "cancer of the rectum" to include "cancer of the intestine" this aptly expresses the situation, since so much depends upon the time, the place, and the surgeon. Let me again warn you that the desire to do an ideal operation should be subordinated to the general condition of your patient.

Before concluding I have still to consider tuberculosis of the intestines, a condition which in many respects resembles malignancy, and occasionally is associated with it. Although as early as 1837 Corbin described tuberculosis localized in the cecum and appendix, and Hallowell, in 1838, reported a death from peritonitis due to perforation of a tuberculous appendix, the subject did not attract the active attention of surgeons until much later. It was only with the development of abdominal surgery after 1880, and as the region of the cecum was opened to light, that surgical removal of tuberculous tumors was undertaken. At first interpreted as a malignant growth, it was gradually recognized that the peculiar circumscribed neoplasm remaining localized for a considerable time was, in reality, a tuberculous process.

Long before Koch's discovery of the tubercle bacillus the question of the portal of entry of the infection occupied the attention of the profession, the discussion centering mainly upon the rôle of milk from tuberculous cattle as the etiologic factor in primary intestinal tuberculosis, especially in children.

According to Ribbert, the coexistence of primary tuberculosis in the lungs and in the intestinal tract is entirely logical, inasmuch as in inhaling the bacillus some of the germs settling in the mouth or in the nasopharyngeal passages are apt to be swallowed with the sputum. The disease may also result from direct infection from pulmonary tuberculosis by means of swallowed sputum, and is characterized by extensive ulcerative lesions associated with diarrhea. This is usually a late stage of phthisis and is not amenable to surgery.

A second less common and less destructive type appears as a localized hyperplastic stricture, associated with constipation. Infection here may take place either through the blood-stream or through the lymphatics. In about half the cases the condition is a secondary one to some primary focus not necessarily in the lungs. It occurs more frequently in the female, the proportion being about 2 to 1. Murphy attributed the preponderance of the disease among women to the fact that the Fallopian tubes are so often the primary site of the lesion. Whether secondary or primary, its most frequent site is in the lower ileum and the ileocecal region. This to a large extent is explained by the presence of the numerous lymph-follicles and by the stagnation of food in the ileocecal region. The appendix and the lymphatics at the ileocecal angle also no doubt play a leading rôle. The initial lesions caused by the bacterial activity are seen in the development of tuberculous nodules in the

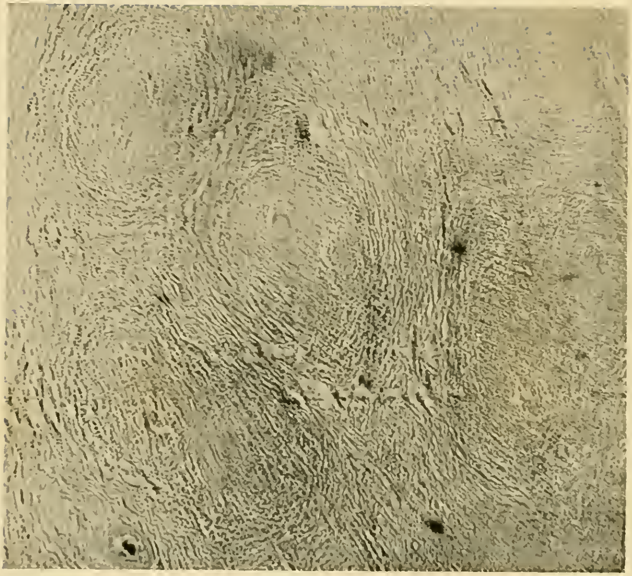

Fig. 23.—Miliary tuberculosis of the ileum. This case was remarkable in that the tuberculous process had produced almost complete obstruction, which was completed after the patient had eaten almost an entire watermelon, seeds and all. The seeds were packed hard against the tuberculous constriction.

submucous and subserous layers, which in spreading, caseate and finally rupture on the surface of the mucosa with the typical ulceration and necrosis as the result. Dissemination occurs by way of the lymphatics along the mesentery of the bowel. It is often said that tuberculosis of the ileum is a local disease. It probably is in its incipient stage, but, unfortunately, the cases do not come to the notice of the surgeon at this early time. Acute perforation occurs in about 5 per cent. of the cases and, as a rule, the process is a rapidly fatal one. The usual result is a chronic infiltration of the bowel and an accompanying production of adhesions matting the coils of bowel together and leading to intestinal obstruction.

The most common site of intestinal tuberculosis is the cecum. In this region and in the rectum massive hyperplastic tumors are built up. The cecal wall becomes thickened and infiltrated, and in consequence of this hypertrophy, stricture and stenosis of the lumen of the bowel may result. The bowel above the stricture tends to hypertrophy and to dilate, while below the stricture atrophy occurs. The appendix may be incorporated in the general mass, or there may be a primary appendicular tuberculosis with no involvement of the cecum or the ileum. In fact, the accidental finding of a tuberculous appendicitis may be the first intimation of the presence in the body of the disease.

Intestinal tuberculosis is apt to be latent for some time. The symptoms at first are merely those of a digestive disturbance accompanied by constipation or diarrhea. But with the advance of the process, the symptoms become those of obstruction—colicky pain, vomiting, constipation, or possibly diarrhea as the result of ulceration or other irritating conditions. The abdomen is sometimes distended, peristalsis is increased, associated with irregular tenderness. Sometimes enlarged mesenteric glands may be palpated. It is seldom, however, that the diagnosis is made before operation unless there is a pronounced history of tuberculosis as a guide.

If the process is localized in the appendix, the symptoms are very apt to be those of a chronic appendicitis, or perhaps indications of typical acute appendicitis bring the patient to operation, when the correct diagnosis is made. In making the diagnosis we must not be misled by the presence of a history of tuberculosis, since appendicitis in a tuberculous individual is not at all uncommon. It is only by pathologic study of the removed organ and the finding of definite tuberculosis that the diagnosis can be established. The tuberculous ileocecal tumor also has a somewhat indefinite train of symptoms—digestive disturbances, such as bloating, belching and anorexia, vague pains, sometimes localized in the ileocecal region, constipation oftentimes alternating with diarrhea. Sooner or later the tumor is palpable in the right iliac fossa. With its progress it encroaches on the lumen of the bowel, presenting symptoms of stenosis suggestive of chronic ileus. Abscess or fistula formation is not unusual.

The main points in differential diagnosis are the history and the prolonged chronicity of the tuberculous process. While the various inflammatory diseases of the intestines may have to be excluded, these do not so often cause confusion as malignancy. Here differentiation is difficult, often impossible, for not infrequently tuberculosis and malignancy are associated. To procrastinate until every symptom is present before making a positive diagnosis is like refusing to diagnose a cancer of the stomach until anacidity, lactic acid, and the Boas-Oppler bacilli are present in the gastric contents.

Sometimes also tuberculosis may develop in a fecal fistula following appendectomy for an apparently simple acute suppurative appendicitis.

The treatment of tuberculosis of the intestines if seen early is oftentimes surgical. Sometimes merely removal of the appendix leaving the affected glands in the ileocecal region undisturbed, may affect a cure. This is, however, not to be thought of as the method of choice, since the disease may continue to progress until at some future time the condition becomes inoperable.

Surgery for tuberculosis of the intestines does not materially differ from that of malignancy, except that there may not be the same necessity for removing enlarged glands as in carcinoma. The prognosis, however, is rather more favorable in tuberculosis. One cannot lay down a law as to whether exclusion or resection is preferable in any particular case. The extent of the lesion and the ease with which radical surgery can be performed must be our guide. It is obvious, however, that resection offers the best chance of a cure. I advise against attempting to separate adherent coils of bowel, since perforation is practically inevitable. In tuberculous disease of the rectum surgical treatment can do little, and if there are numerous abscesses and fistulæ associated with rectal hemorrhage, I advise colostomy. The patient is made much more comfortable and the secondarily infected tuberculous ulcerations can thus be better controlled. In all these cases, if operation is decided upon, I prefer to do it under nitrous oxid and oxygen anesthesia.

The benefits of surgery in other types of surgical tuberculosis are well recognized, and manifestly illustrate that with the removal of the focus of infection we may logically expect a rapid elimination of the effects of the infection leading to a cure.

Before closing I want to thank my hearers for their indulgence. I am aware that it might have been better to have chosen one subject and have developed it exhaustively in the course of these lectures. But at the suggestion of your committee I concluded that a series like this might be more interesting to you. I feel that I have in a rather bold and perhaps imperfect outline merely given the outstanding features of the topics discussed. The main deduction to be drawn from them is that it is folly to attempt to treat medically conditions which manifestly belong to the domain of surgery, and when they are threatened as they are by an overwhelming array of the enemy forces, aid from the outside is imperative. The time may some day arrive when less drastic measures than surgical incision may be available, but that day it seems to me is still far distant. And, in the meanwhile, we have every reason to be grateful that with the foundations laid by Long, Morton, Pasteur, and Lister we have been able to develop surgical methods which can and do overcome many of the destructive forces that invade the human body.

Pathology

The surgeon is not the only one to whom the appendix is an object of extreme interest. The pathologist also has his problems with this organ, and while many questions have been answered more or less satisfactorily, there are still many more which require solution. If we begin by considering the pathologic anatomy of the organ, we see that of all morbid processes inflammation is by far the most important. Inflammation may be considered to begin ordinarily in the mucosa and possibly down at the bottom of crypts. Ulceration begins quickly and the walls of the lumen are covered by a membrane of fibrin, pus-cells, and mucus. Extension into and through the walls is very rapid; grossly, the organ becomes stiffened and thickened, and exudate, usually fibrinous, appears on the serous surface. Histologically there is general congestion, edema, infiltration of pus-cells through all coats with a plastic exudate of fibrinous purulent material on the serosa. Localized abscesses may develop; gangrene may supervene with perforation and softening of the entire organ. Thus far the conditions are plain enough, but when we ask the question "does infection come always from within or does it come occasionally via the blood-stream?" we are confronted with a real problem. The experience of the majority of patients with acute appendicitis is that the condition is rather sudden in its onset, and the entire body takes part very quickly as manifested by marked feeling of ill health, chilliness, the generalized abdominal pain, and perhaps vomiting. One cannot say that there is any organism which is the specific cause of appendicitis. There are certain ones, however, which are found more often than others. Can they come from the blood-stream electively, choosing the appendix, or must they enter from within the lumen? If they come from the blood-stream and electively enter the appendix, is there something peculiar about the appendix itself which determines it, neglecting for the moment the elective affinity of an organism—a much more difficult question. Why not some other portion of the intestinal tract? A short consideration will lead to the realization that the appendix is a peculiar anatomic structure. It not only has a blind end and a narrow lumen but also a very rich lymphatic apparatus. In regard to its blood-supply, a number of observers believe they have found an appendicitis beginning immediately around some small terminal arteries. If the subject is examined experimentally, we find in animals that it is possible to produce an appendicitis practically only when some means are used to occlude the lumen. The human appendix is notorious for its changes of position, for its adhesions, kinks, and constrictions, all of which assist in obstructing its outlet. The idea that obstructed contents are more dangerous has often been expressed; not so much in relation to an increase in the number of bacteria; . . . we know, of course, that this is the most heavily infected part of the intestinal tract, . . . but more in relation to their virulence or to the production of more powerful toxins. Referring again to the fact of its vascular supply, it has been found that certain vasoconstrictor substances are present in stagnating appendical contents, which has led to the statement that perhaps by constricting vessels this favors invasion by organisms, but we need more data in regard to these substances in such stagnating contents before such a rather tempting theory can be seriously entertained. Our own cases of chronic appendicitis have been habitually constipated in the great majority of instances. The cause and effect are not clear, however, but from some of the succeeding remarks it will be apparent that the question is of distinct importance. There is one very important fact in relation to its blood-supply, and that is, its connection with the portal system. Infection may reach the liver by this route. In the acute variety we have found in those cases in which the gangrenous and suppurative processes, especially the former, have involved the meso-appendix to any extent, that the danger of ascending suppurative pyelophlebitis with its multiple abscesses in the liver and its inevitably fatal result is to be entertained with the greatest respect. In the chronic variety, assuming for the moment that toxins are really formed and absorbed, and likely enough they are, the liver may be continually irritated by its endeavor to render them harmless. Failing in this work, the toxins may engender constitutional symptoms, irritation of the kidneys, etc. When bacteria also enter the veins of the meso-appendix—we know that bacteria may penetrate even an intact bowel wall—they may directly involve the bile-passages after their excretion by the liver, as pointed out in the article on the gall-bladder and bile-passages. As to its lymphatic relationships, that the appendix is not innocent in originating lymphadenitis or lymphatic extension to the gall-bladder, stomach, and pancreas, has been emphasized for many years not only by Dr. Deaver, but by many others.

Turning to the question of chronic appendicitis, we are immediately confronted with the question of what shall be called "chronic appendicitis." From the pathologic side we conceive of a normal appendix as having the same caliber throughout, tapering very abruptly to a rounded tip. The organ should appear steely blue, with a few vessels showing through the smooth transparent peritoneal surface; but most appendices show an unevenness in their caliber, there are opaque spots or areas, many show distinct thickening of the peritoneum, with considerable congestion of the vessels. Microscopically it can be shown that there are deposits of scar tissue, very seldom even in distribution, but more often in foci of greater or less extent. By a chronic inflammation in the strict sense of the term we understand a progressing process, characterized by continuing deposits of connective tissue and lymphocytes. In these appendices, as well as in other organs, it is impossible to determine from the specimen alone whether the connective tissue is a mere scar, the result of healing of an old inflammation, whether it is a progressive chronic inflammation, or whether it is simply the connective-tissue deposit of advancing age. There are some who believe that all scar tissue in the appendix is a result of repeated acute inflammations, however slight, but we believe this cannot be correct, and in order more satisfactorily to judge of these possibilities, we must consider not only the appendix, but the patient from whom it was removed. From our histories we can readily separate those individuals who have had one or more undoubted sharp attacks of acute appendicitis from those with so-called chronic appendicitis. The symptoms presented by the latter are usually ill-defined and vague, consisting of odd pains, never very severe, in the right iliac fossa, together with some tenderness and aching, perhaps with "indigestion." The only symptom which we have found quite consistently is constipation. If we turn to the results, as found in our follow-up clinic, we discover that about 60 per cent. of these patients are cured, that is, they are relieved of the immediate symptoms of which they complained. With these factors in mind, we have attempted to correlate the pathologic anatomic findings, but have arrived at no certain conclusion.

Those patients who had complained of symptoms, which, after careful study were diagnosed as due to chronic appendicitis, and in whom the symptoms were relieved by removal of the appendix, did not have appendices much different pathologically anatomically from either those whose appendices had been removed for chronic appendicitis without relief of symptoms, or those from whom the appendix was removed incidentally. There is one thing, however, which seems to be shown, and that is that the patients who had upper abdominal symptoms with chronic appendicitis quite often had a concretion in their appendix. Among the foreign bodies we have often found the oxyuris vermicularis, but here again we are in no position to state any positive conclusion from this finding.

There is a very interesting tumor which arises in the appendix and which anatomically has all the characteristics of a carcinoma. In our routine, in which all specimens are examined both grossly and microscopically, it has occurred in from 0.2 to 0.4 per cent. of the specimens; it has never been diagnosed clinically, the appendix having been removed for either acute or chronic inflammation, or incidentally during the course of another operation; it has been recognized in the laboratory grossly in only about half of the instances. Its peculiarity lies in its localization to the appendix with every histologic evidence—in a limited field—of invasive tendencies, that is, if a field were chosen in the midst of the growth, and any pathologist were asked concerning the diagnosis, he would have no hesitation in saying that it is a malignant tumor. It occurs in two main varieties: The first, similar to tumors arising in other parts of the intestinal tract in which the cells are columnar, and arranged more or less "adeno" fashion, and the second, in which the cells are smaller, polygonal, or spheric, and arranged in clumps and nests.

Connective tissue is usually sparse in the first variety, but quite often the entire tumor is surrounded by a definite capsule. Reports have shown that mucoid and gelatinous material is formed sometimes in large quantities. In several of our specimens most of the tumor cells had been destroyed by this material. They are very similar histologically to carcinomata arising in other parts of the intestinal tract, and it is said that the age incidence is the same (fifty-two years—Ewing), but in our 4 cases the ages were eight, nineteen, thirty-two, and thirty-four years, respectively. There has been no recurrence in 2, concerning whom we have data, for eight and two years (the cases aged eight and thirty-two years respectively, at operation), but in the literature it is found that it is the columnar cell type which, as a rule, recurs and produces metastases, although this event is uncommon (6 per cent.—Batzdorf). In our specimens, which now number 25, we have several examples in which the growth has penetrated and appears in the serosa; in all the others the tumor extends no further than the outer layer of the muscularis.

The second or polygonal cell variety is characteristic of the appendix and resembles the carcinoma of no other portion of the gastro-intestinal tract except, perhaps, the small multiple tumors of the ileum reported by Lubarsch, Bunting, and others (see Ewing, Neoplastic Diseases). Here then, is a tumor which anatomically is a carcinoma, and which clinically is benign; what shall we say of it? There is but one evidence which consistently led to the suspicion of the presence of the

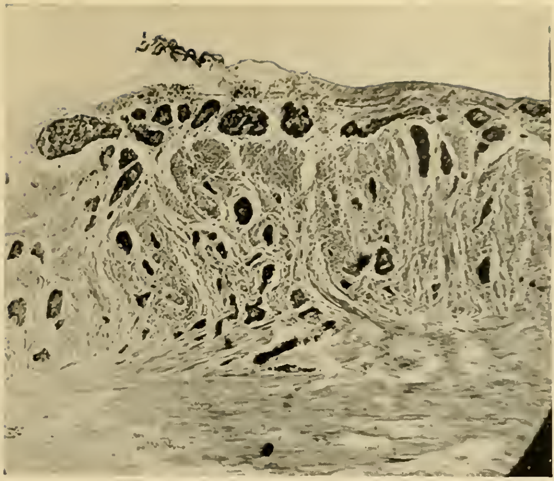

Fig. 24.—Primary carcinoma of the appendix. The dark masses are groups of small tumor cells which have infiltrated through the muscularis and through to the serosa. This is the only one of our series of 28 cases in which such infiltration into the serosa was demonstrated.

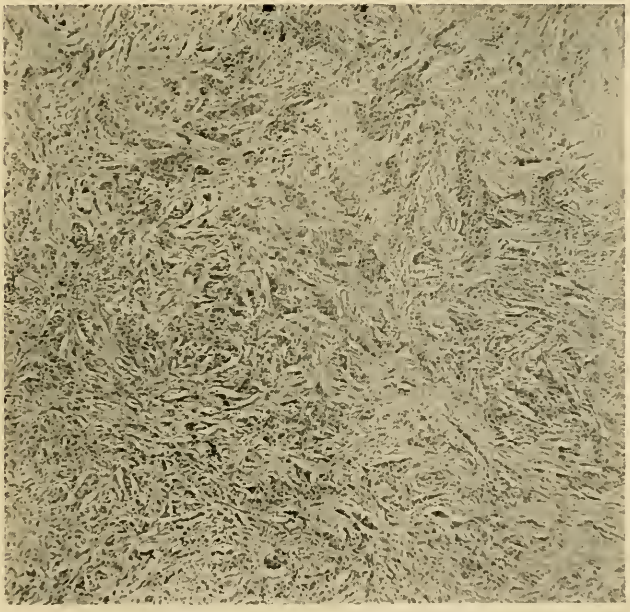

Fig. 25.—Small cell primary carcinoma of the appendix. This tumor was a circumscribed nodule, roughly globular, about 0.6 cm. in diameter, occupying the position of the lumen and the submucosa at the distal end. No infiltration beyond the submucosa.

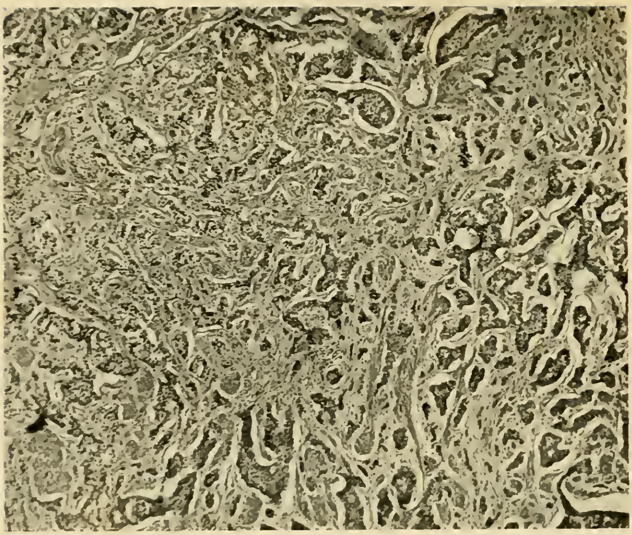

Fig. 26.—Primary carcinoma of the appendix. Section from the position of the lumen of the appendix as in Fig. 24.

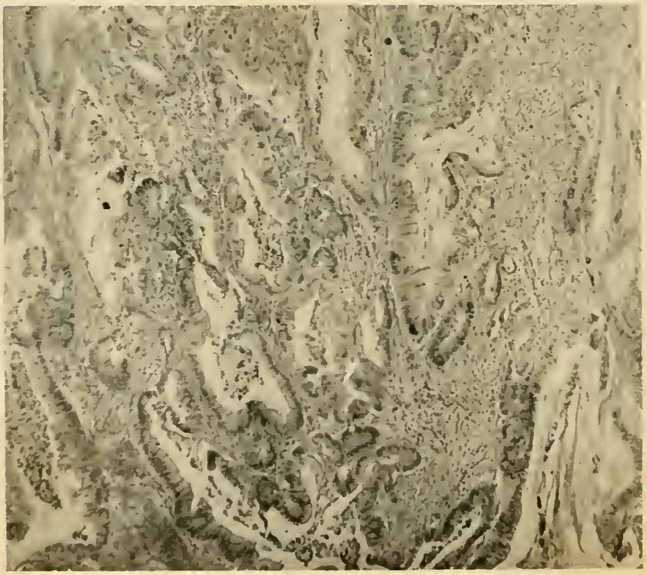

Fig. 27.—Adenocarcinoma of the rectum.

tumor in our specimens, and that was a clubbed distal end. We have had it occur, however, in all parts of the organ, but in the great majority it was this distal clubbed end which showed the tumor. This tumor, therefore, in an atrophying organ seems very interesting, and it is important that every case be carefully watched postoperatively, and any of these cases which give suspicion of recurrence or metastasis should be thoroughly studied.

Dr. Deaver has chosen as one of his other subjects carcinomata of the intestines. He has mentioned the fact that of the malignant tumors, this is by far the most common. In our records of 135 specimens sent to the laboratory in the last fifteen years, 125 were carcinomata and 10 were sarcomata. The treatment for these tumors, if they are discovered at a favorable time, is, of course, resection, and the question naturally arises: How much can be removed, and what disturbances result? We know that the entire colon can be removed without very great danger to life, but there is disagreement as to the comfort of the patients on whom this operation has been performed. Since, however, the small intestine is by far the more important organ in digestion and absorption, it is naturally of more interest to inquire into the results of resections of this portion of the gut. True it is that tumors of the small bowel are far less frequent than those of the large intestine, but resections are often required, if not for tumors, then for some other reason. In our particular series of tumors there were 15 in the jejunum and ileum; 5 in the duodenum; the remainder being divided in the large intestine as follows: 12 in the cecum; 26 in the colon; 25 in the sigmoid, and 52 in the rectum. Of the pathologic anatomy little can be said by us which has not been recorded many times over. We have encountered them in the ordinary varieties of adenocarcinoma, mucoid carcinoma, carcinoma simplex and scirrhous, with a few polypoid tumors, and a few showing large cysts with papilliferous ingrowths.

The choice, where a large segment of intestine is to be resected, does not ordinarily rest upon the free will of the operator, but must vary with the exigencies of the particular case. The total length of the intestine in man varies somewhat, with the mean as given in a set of measurements of 5.6 to 8.7 meters, so that in considering the subject the question is not altogether one of the absolute length which is removed, but rather the ratio of the resected portion to that of the part remaining. Reports of cases have indicated that about one-half of the small intestine may be resected without endangering the subsequent life of the patient. Whether resection of the jejunum or of the ileum is the more dangerous has been answered in both directions by an almost evenly divided number of writers, and it may be assumed that in all probability the difference between the two is not very great, although with few exceptions the extensive resections in humans were of the ileum. It may be interesting to recall the case described by Ruggi in 1896, in which a review of the literature can also be found (Policlinico, 3/1896), in a boy eight years of age who had had repeated intestinal obstructions following a wound. Ruggi resected a large mass of densely adherent, knotted gut. The part removed measured 3.3 meters, the lower part having been removed at 15 cm. above the ileocecal valve; obviously the upper part must have been in the jejunum. The boy suffered very violent

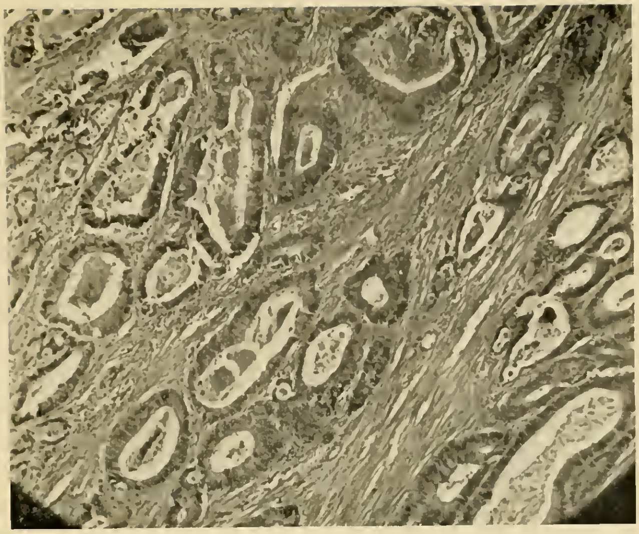

Fig. 28.—Adenocarcinoma arising in the cecum.

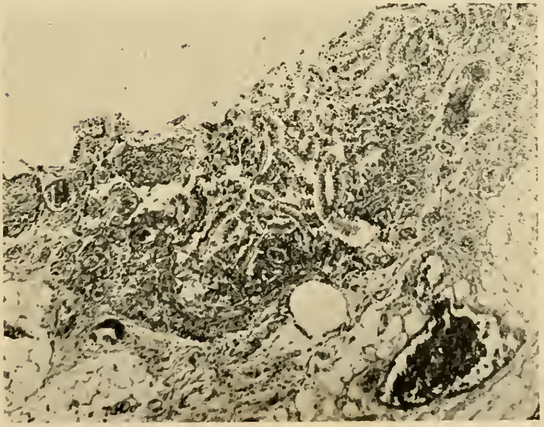

Fig. 29.—Adenocarcinoma of the cecum showing a group of the tumor cells in the thickened serosa of that organ.

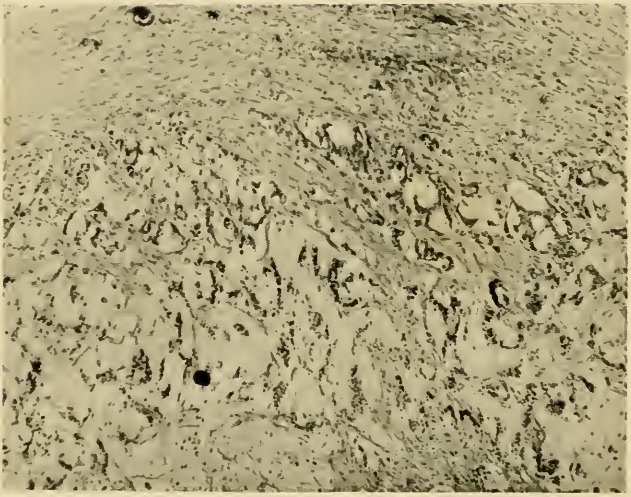

Fig. 30.—Mucoid carcinoma of the rectum. The clear spaces, in which the dark stained tumor cells appear, contain mucus.

hunger which lasted for almost a month, when his general nutrition began to improve decidedly. A metabolic study (not as complete as could be desired) showed that the patient was utilizing his food in a remarkably efficient manner. In three months the loss of weight due to the disturbed nutrition from obstruction had been entirely compensated. This case illustrates the very remarkable adaptability of the digestive tract, an adaptability which is greater than we would suppose *a priori*, and one without which surgical methods would be totally impossible. But this last matter is one which applies to all parts of the body and need not be developed any further in this discourse. If we examine this adaptability a little more closely, we find from the studies of various observers both on human subjects and on animals, particularly by means of fistulæ in the latter, that after resection of the ileum there seems to be an increased secretion of gastric juice and a delayed evacuation of the stomach. Intestinal movements are also slowed and the cleavage of the food is carried further in the jejunum. Many of the dogs, however, from which large amounts of ileum were removed, died in a number of months with diarrhea and general loss of strength and weight. When the resection is on the borderline of the possible, metabolism determinations have shown that utilization of carbohydrates is good; that of proteins is impaired although sufficient, but fat utilization is very poor. The practical deduction to be drawn is that abundant food rich in nitrogen must be supplied, meat being the best. It is inadvisable to increase the intake of fats, and those that are given should be of a nature easily absorbed, such as olive oil, lard, etc. Carbohydrates can be given abundantly, especially since they spare proteins.

The anatomic findings following resection were not uniform. It is said that hypertrophy and hyperplasia of the mucosa occur, but others could demonstrate no anatomic changes, and for the present this question must be left open.

THE CONTRIBUTION OF PASTEUR TO MODERN SURGERY[1]

This is a day of thanksgiving as well as of commemoration. We have come together from all walks of life, from all branches of science, and from all parts of the country to give praise to an immortal. Today we are in such a state of specialization that the work of each of us is confined to a strictly limited field. The assemblage, therefore, of so many specialists at this time, each eager to tell of the contributions of this one man, this genius, whose centenary we are celebrating, to his own speciality, is more full of meaning than that which any or all of us can say.

"Chance favors only the prepared mind." These words of Pasteur, uttered as a parenthetic phrase in one of his lectures to his students, are significant of the habit of thought and the career of the man whose researches so vitally affected scientific thinking and the scientific activity of his time. While this characteristic of bringing the prepared mind to the study of a subject is an acquired one, when, as in this instance, it is combined with innate qualities, such as imagination, unwavering enthusiasm, accuracy of observation, logical deduction, independence of thought, and, above all, scrupulous honesty and an inordinate capacity for work, it represents the very essential of greatness.

I can think of no other scientist whose work has been so far reaching and who has contributed to so many divergent activities of modern science. We are here to do honor to the man who laid the foundation of bacteriology and immunology, of modern aseptic surgery, and of modern medicine in some of its most important aspects. Chemistry, physics, agriculture, and many of the industries owe to him an incalculable debt. Does not the enumeration of this varied list of debtors indicate the fundamental nature of the discoveries of Pasteur? No scientist, medical or otherwise, in the annals of human endeavor has produced such lasting benefits for the health and well-being of mankind.

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[1] Delivered at the Pasteur Centenary Celebration, Philadelphia, December 27, 1922.

His life emulated that of the ideal research worker, for not only was he able to carry out his investigations into the theory of his problems, but he realized the value of the results and perceived the practical application of them.

His researches began in the field of chemistry, and in this field he made the discovery of the molecular dissymmetry of tartaric and paratartaric acid. By definite steps of logical deduction and accurate observation he went from his first discovery to the fundamental one on which his later contributions depended, that is, the overthrow of the theory of spontaneous generation, and the substitution therefor of the doctrine that all life proceeds from life, or, as he expressed it, "life is the germ and the germ is life."

In 1856 Pasteur began a long series of experiments which conclusively proved that for each form of fermentation studied by him there was one specific cause and one only. This incontrovertible proof of the rôle of the germ life in the processes of fermentation and putrefaction represents the turning-point in scientific reasoning, scientific research, and the practical application of scientific prinicples. Pasteur's researches along these lines began with a study of the various diseases of French wines and beer. How fortunate that Volsteadism was not, and let us hope never will be, active in France.

Not only were the early investigations of Pasteur important to the brewer and the wine merchant, but they were of vast monetary value to the silk industry. Never in his long life did he admit the possibility of enriching himself from his discoveries, but through them France was enabled to pay off the heavy indemnity imposed upon her following the Franco-Prussian War. Therefore, as Chambrun has said, "his work had become a national possession." It is significant of the vision which characterizes the thinker, that while still on the threshold of his successful demonstration of the truth of the theory that fermentation, as well as putrefaction, was an effect of life, Pasteur saw the analogy between fermentation and disease, and even hoped that these studies might be pushed far enough for a serious investigation into the origin of various diseases. From this pious wish there ultimately resulted what may well be considered Pasteur's second greatest achievement, that of immunization against infectious disorders, forming as it does the basis of modern methods for the control and possible eradication of certain diseases.

Pasteur's work in this field was not done without having to overcome obstacles thrown in his way by ignorant people who could not realize the extent of the mischief they might have done had their plans succeeded in preventing Pasteur's use of animals for experimental work. How would and how can the boundaries of surgery be broadened if our work is to be hampered by legislative restriction? Pasteur, like those who preceded and followed him, was opposed and decried, instead of being encouraged and helped. For, unfortunatley, the politician sees no votes in experimental research and the layman rarely appreciates its value.

Not only were his proofs of the existence of specific transmissible microbes questioned and the neccessity of animal experimentation, in order to isolate and find modes of destroying these organisms, denied by the layman, but even the medical profession of France received his work with incredulity and lack of interest. It is unfortunate that the medical profession was unable to discern the truth. Notwithstanding their hostility and opposition, Pasteur carried on his work, demonstrating even more fully by his results, in the diseases of anthrax and rabies, the correctness of his theories. The truth is mighty and will prevail.

The generosity of this truly great mind is evident in the fact that during the progress of his painstaking and interesting endeavors Pasteur, instead of coining a new word for his protective injections of attenuated virus, chose the term "vaccination," which had been used by Jenner, because of its derivation from *vacca*, the cow. In thus acknowledging his indebtedness to Jenner, the originator of this preventive measure against smallpox, Pasteur, in addressing a meeting of the International Congress of Medicine in London, remarked, "I have expanded the meaning of the word 'vaccination' which I hope will meet the approval of science, since I wish to pay homage to the tremendous service rendered to humanity by one of England's greatest men—your Jenner. Indeed, I deem it a special privilege to honor his name in this noble and hospitable city of London."

What surgery owes to Pasteur is the firm foundation which made possible the great edifice of modern surgery, still in the process of erection, but which is now showing its real magnificence and noble concepts. On this foundation, laid by Pasteur, Lister was able to begin the erection of the superstructure of aseptic surgery. In 1874 Lister, writing to Pasteur, said, "If you come to Edinburg, you would be rewarded by seeing at our hospital in what full measure your labors have benefited the human race. Need I add what a great satisfaction it would give me to show you here what surgery owes to you." What a joy this tribute from a master surgeon and scientist must have been to Pasteur, surrounded as he was by those who seemed blind to the truth.

The great evils of the nineteenth century in the field of surgery were pain and infection. After the year 1846 we find the introduction of anesthetics to abolish pain becoming general. This completely revolutionized the possibilities of the application of surgery to diseased processes. However, the hope that the surgeon, with the abolition of pain, might extend his field of usefulness, beyond a few minor operations and emergency amputations was hampered and frustrated by the all too frequent occurrence of infection with all its disastrous consequences. Occasionally a wound healed primarily, without the formation of pus, but the majority became "putrid," to use the then current phrase, or, as we say, infected. It is impossible for me to even attempt to portray the horrors and mortality attached to surgical procedures at the period when Pasteur was conducting his researches. Pus poured from the wounds, and hospital gangrene, erysipelas, pyemia, and septicemia were stumbling-blocks to all surgical progress. The risks of entering a hospital were so great that the surgeon's hands seemed indeed to be paralyzed by the fear of hospital disease, "that blind Nemesis that presided over their work." Operations performed in private houses gave much better results than those in old hospital wards with their overcrowded, unsanitary conditions and accumulated infection. Even the cleanest hospitals of that time were hotbeds of infection; patients with suppurating wounds were crowded in a single room without any precautions to protect one from the other. In fact, the conditions were ideal for the transmission of infection from doctor to patient, and from patient to patient. It is difficult for this generation to realize the status of hospital technic in those days and the changes that have been wrought since then. A few words may suffice to give you a slight idea of the change:

In those days any room was used as an operating-room and any old coat, the older apparently the better, served the surgeon as an operating gown. Sometimes a linen duster was the garment chosen, and when a young man actually discarded his coat and worked in his shirt sleeves, he was looked upon as a bold innovator. Sponges, so-called surgical sponges, used during the operation were of better texture than ordinary bath sponges, and when of good quality they lasted a long time. They were used over and over again and sometimes had even served for removing blood from the table and perhaps also the floor. After being washed in ordinary water they were placed uncovered in a bag along with the surgical instruments, ready for the next victim. Assistants at the operation stood around without regard to protecting the patient from infection carried on their clothing or their hands. Infection was not, as today, a disgrace and an exception, but almost the invariable rule. No wonder that going to a hospital meant leaving all hope behind.

When, therefore, Lister, who, had been working long in Glasgow on the problems of inflammation and the decomposition of wound discharges, read in 1865 of the work of Pasteur, he realized that the results were applicable to surgery. Lister read Pasteur's assertion that "the most far-reaching of my researches is simple enough; it is that putrefaction is produced by living ferments." In this same dissertation Pasteur differentiated between aerobic and anaërobic infections.

Pasteur threw light on what had hitherto been darkness, and Lister, with the mystery dispelled, wrote in 1869: "When it had been shown by the researches of Pasteur that the septic property of the atmosphere depended on minute organisms, it occurred to me that decomposition in an injured part might be avoided by applying as a dressing some material capable of destroying the life of the floating particles."

This marked the birth of antisepsis in surgical procedures, and was the culmination of the unceasing labors of the French scientist, conferring one of the greatest if not the greatest boon ever received by suffering humanity.

With this pronouncement of the principle of antisepsis and its subsequent development into that of asepsis, the main barriers in the path of surgical advance were thrown down. With a phenomenal pace the onward march has continued, and at this time there is practically no part of the human body closed to the aseptic knife. The surgeon has advanced from the limbs to the trunk, from the abdomen to the cranium, from the cranium even to the heart, until it seems as if the Listerian knife were to prove the long-sought panacea.

There naturally followed also a complete revolution in operating-room technic and hospital conditions. The words "surgically clean" now imply the acme of cleanliness to be attained only in a modern hospital. The ordinary wash-basin has given way to porcelain-lined trays, sea-sponges to gauze used but once; the bone or wooden-handled knife to the one-piece metal instrument. The protection of the patient is the prime consideration; doctors and nurses are clothed in immaculate sterile gowns, with masks covering the head, the mouth, and the nose. The operation itself is conducted in a well-lighted clean room, with sterilized instruments, and the operator's hands are encased in sterilized rubber gloves. The surgeon works with a reasonable certainty of success not born of arrogance, but with the knowledge derived from the application of the scientific principles of protection as formulated by Lister, based on the work of Pasteur. The emblem of despair no longer darkens the door of our hospitals, but has been replaced by that of fully justified hope for restoration to health and vigor. No longer is the mortality of amputations from 40 to 50 per cent. While Lister in 1865 wrote that in 15 cases of excision of the wrist-joint by his method 6 patients developed hospital gangrene and 1 died of pyemia, today these complications are the curiosities of the surgical wards. The term "hospital" itself may be said to have resumed its original meaning of a hospice or haven for the tired, stricken traveler along life's journey. Hospital treatment instead of being shunned is eagerly sought by poor and rich alike; indeed, the supply scarcely meets the demand.

In no branch of medicine and surgery are the beneficent effects of this revolution more apparent than in the protection afforded the expectant mother in her hour of supreme trial. As has well been said, with his antirabic inoculations he conserved the child for the mother; and with his demonstration of the cause of puerperal fever he conserved the mother for the child. The frightful mortality from so-called epidemics of puerperal or child-bed fever is happily a thing of the past, because Pasteur discovered the minute organism which caused the fever. It is related that at a meeting of the Academy of Medicine of Paris, during a discussion on the subject of puerperal fever, Pasteur interrupted the remarks of one of the most eminent physicians of his time by boldly calling out from his seat: "You are entirely wrong as to the cause of these epidemics; it is the doctor and his assistants who carry the microbe from a sick woman to a healthy one." The speaker replied that he scarcely thought this microbe would ever be found, whereupon Pasteur jumped to the blackboard and drew the now familiar figure of a chain of streptococci, saying, "there it is, that's what it looks like." Pasteur discovered the cause and Lister applied the remedy, and the safety of motherhood has been practically assured.

What surgery also owes to Pasteur and to Lister is the extension not only of the operative field of the surgeon, but the expansion of his knowledge of diseased processes; that is to say, the opportunity of observing a given disease in its various stages. Hitherto the practice of surgery had been based upon the knowledge obtained from the study of clinical symptoms and of the terminal stage of disease as seen in the postmortem chamber.

The latter, as we know, led to incorrect deductions due to false impressions, resulting from the changes produced by death. These inaccuracies we are now able to replace with the more accurate observations on the living subject in the operating amphitheater. For example, it certainly is of more value to see the precise conditions which cause, let us say, a gastric trouble, at the time of the illness, than to observe the same trouble some time later when perhaps other changes during life and after death have been added to the pathologic picture. In order to heal the living we need the knowledge derived from living processes. This knowledge, when correlated with clinical symptoms, necessarily is reflected in our greater ability to make a correct diagnosis and consequently in improved methods of treatment. For, instead of treating only symptoms, we are now in a position often to focus treatment directly on the basic cause of a disorder. And in this way surgery falls in line with preventive work, the watchword of our times, which in the last analysis represents the ultimate triumph of Pasteur. If, then, his work is to be judged by his contributions to humanity, Pasteur will and should rank with the greatest of the immortals, for his has been the guiding hand in the abolition of the surgical disasters of our forefathers.

Mr. Stephen Paget admirably describes this immortal figure in the history of the world: "To change the whole outlook of medicine and surgery, Heaven took and trained a 'pure scientist,' who had never done an operation nor written a prescription; a man who had to screw up his courage even to look at some of the ordinary sights of a hospital; took this non-medical man of science and set him to be head of all the heads of the medical profession, to have them all obedient to his teaching, and proud of the very sound of his beloved name. . . . The whole world is well aware that he has availed more than the physicians and surgeons of his time for its health and happiness. He was set apart from them, that he might be the leader of them; and he led them into that kingdom which they longed for, but could not have found for themselves."

And so on the centenary of his birth and the twenty-seventh anniversary of his death we have gathered here to say words in simple tribute. But words are powerless, while the child of his genius is still nobly testifying to its truthful conception and fulfiling the highest hopes of an enlightened and enriched science.

MEDICAL EDUCATION AND EDUCATORS

Complacency and smug satisfaction are danger signals of decadence, just as wholesome discontent and healthy introspection and self-criticism are indications of the will and desire for improvement. It is on the basis of the latter that we are constantly seeing changes in almost every branch of human endeavor. Whether or not these changes are always synonymous with improvement is a matter of debate, particularly when they concern our methods of education, general and special. There is, in fact, often a great deal of criticism hurled against the lack of stability and the continual changes that are going on in our general educational system throughout the country. Diversity of opinion exists not only as to the best methods of imparting knowledge, but even more so as to the actual essentials of a modern education. There are those who would banish everything that does not directly pertain to the practical, and there are those who lay more stress on the cultural studies as the proper preparation for life. This general unrest naturally extends to the higher schools and to special schools, such as the medical college and university. With regard to medical education the most marked changes that have been produced by the widening of the field of medicine compared with that of yesterday are primarily the increased requirements for entrance to a medical school, and secondly, the manifold subjects taught within its walls. Of these two fundamental changes there is little doubt that the first mentioned is universally accepted as wholesome and beneficial to the status of the profession. The young man entering the medical school with a preliminary college education including a year or two in premedical work in the biologic sciences comes with a mind prepared for at least some of the problems he will encounter throughout his medical studies. There is, however, less uniformity of opinion as to the advantages of the increased number of subjects included in the curriculum of most of our medical schools. The question naturally resolves itself into one of the true functions of a medical school, that is to say, whether it aims to train practical physicians or scientific investigators. It is, of course, self-evident that one is as indispensable as the other for the proper functioning of each, but is not scientific research a specialty more suitably provided for in a postmedical course?

The crying need of our day is for practical men who by their training are able to depend as much on their own acumen and powers of observation as upon the various more or less valuable diagnostic and therapeutic adjuncts. The criticism is often, indeed, too often, heard that our medical schools have so overstressed the combination of laboratorian and practitioner as almost to obliterate the latter in the admixture. That is why there is the dearth of doctors, which we hear of throughout the country, and which I believe is especially keenly felt in the South.

If we wish to see the pendulum swing again toward the more practical side of medical teaching the question naturally arises as to the types of teacher best suited for the work. This question has received added stimulus within the past decade or so with the introduction of what has become known as full-time professorships for the practical chairs of our medical schools. These chairs do not differ from others in that their fundamental requirement is the ability to impart knowledge and to advance it. The next essential is a mastery of the subjects to be taught based on practical work within the hospital as well as outside of its walls. Such mastery is possible only after long practical training and can be maintained only by the constant application of the knowledge acquired. It is the maintenance of this knowledge and constantly increasing it that is most vital and essential. And in order to keep it at its highest level the teacher requires the stimulus of the active pursuit of his profession among all sorts and conditions of laymen and the meeting in consultation with all sorts and conditions of doctors. One aspect of the advantages of extrahospital service for the clinical teacher has been happily emphasized by the late T. C. Janeway, whose experiences both as a part-time and full-time professor lend particular value to his opinion. Janeway stresses the point that the services of the clinical professor, by virtue of the talents, knowledge, and experience which presumably have won him the distinction, are most often sought in consultation. This, of course, must nearly always take place in the home of the patient. In addition to the beneficial effects of expert opinion on the patient, much is to be said also of the effects of the same expert opinion on the attending physician. Many a valuable and impressive lesson derived from such consultations has entirely changed the outlook and the prospects of many a general practitioner. If the object of the medical school is to broadcast and advance medical knowledge, why should this advantage be confined to the medical student *per se*. Why not permit it to be extended to the man already in the active world. On the other hand, the value to the teacher of obtaining the viewpoint of the practitioner and realizing his needs is of no less importance, and must naturally be reflected in his class work. It must, of course, and I doubt not that it can be taken for granted that the part-time professor will never allow his outside interests to clash with his college work. Speaking from personal experience I can say, and can say it without laying any flattering unction upon myself, that my clinical teaching always has had the first and paramount consideration in the day's work. I can scarcely believe that this is exceptional, so that aspect of the subject need not enter into the discussion.

From a practical point of view, that is, with regard to remuneration, there are few institutions so well endowed as to make adequate recompense for the relinquishment of the gains of private practice of the expert clinician and surgeon; and few men are so fortunate as to be in a position to relinquish the same. Limiting the professor's activity to his university work may present the temptation of making of the hospital a source of income from private patients, and thus practically divide his attention between the two. And even where the honorarium attached to a full-time professorship is sufficient to meet the requirements of the incumbent, limiting his activity to the university and its hospital is apt to engender a dull routine, stifling to initiative and production.

Here, again, there enters the question of the true function of medical training. Is it to be preparation for the art of practice or for the science of research? It must be remembered that medicine is only about one-tenth science, the other nine-tenths is an art to be acquired by wide, long-continued experience in the world as well as in the hospital. It is doubtful whether the young man trained by a master whose viewpoint is limited to the hospital will, in turn, become as masterful as one who is led by men whose outside contacts give them the broad practical outlook which the art requires. Up to the present time full-time chairs are still being filled by really practical men, but will the future still be able to provide such men under the full-time régime?

On the other hand, is it essential for the clinical professor to devote all his time to hospital practice? If he is the practical and efficient personality which he should be, he will certainly possess the proper sense of the value of teamwork among his staff. Imbued with the importance of training his young assistants in self-reliance and independence of action he will delegate to them the daily routine and the less intricate problems of diagnosis and treatment; the service of the master himself should be required only for supervision and consultation in the more unusual and serious cases. In this teamwork there also enters the interplay of art and science. This will be engendered by the artist in the ward directing the scientist in the laboratory by calling attention to practical and essential problems of research presented by the problems that arise. These suggestions will gain in value by the wide experience of the clinician, who, as has already been remarked, by his contact with the practitioner, is better able to realize the problems of the latter than one who lacks such contacts.

In much of this discussion as to the pros and cons of the full-time clinical professorship the layman's point of view is often overlooked. There is little doubt that every person, intelligent or otherwise, when sick wants to be treated by a man whose practical experience has made him a master artist rather than one well versed in scientific principles, who endeavors to apply them to his patients without regard to the personal needs of the individual concerned. There is no other profession in which the personal elements play so vital a rôle as in the practice of medicine, and where practical results are obtained as much by the power of moral suasion supplemented by the imagination as by the application of rhyme and reason. Even though man has been created free and equal as far as the pursuit of liberty and happiness is concerned, he has not been created equal with regard to his physical and moral needs and requirements. Each individual is a law unto himself in this respect, and the man who knows how to vary the application of his principles to suit the individual is the one who will become the artist supreme and obtain the best practical results. The sick man wants to get well, and he is going to the person who will help to make him well or, in modern parlance, who will deliver the goods. It is for this reason that the -pathies and -isms of today are flourishing. While our laboratories are filled with research workers and the too-scientific doctor is studying his case, the chiropractor and his like are curing the patient, or at least persuading him that he is cured. We can only fight this vicious extraprofessional activity by increasing the supply of well-trained general practitioners, trained by practical men in the practical chairs. This is the true function of the medical school. The training of the scientific worker is a matter of postmedical and even postinternship concern. For I am persuaded that the practical work of intern experience is a fundamental requirement for the development of the best type of medical scientist. Unity of purpose is the distinctive feature of the medical school, and that purpose is the practice of the art of healing the sick.

Finally, it must be remembered that the situation of the medical school and hospital must needs have something to do in the decision of full-time professorships. If it is situated in a small town, where clinical facilities are not large, there is a greater problem to face than if the hospital has a very large population from which to draw. The distinction between these two extremes is, of course, obvious, and while it may be possible for men of very exceptional ability to draw patients, no matter where they are situated, this is not ordinarily the case. The sum and substance, however, of the whole argument can be made clear in a few words, The healing of sick people is an art, and to acquire that art requires enormous experience, and to acquire the enormous experience demands, as we have stated several times, the contact with sick people in all walks of life and under all circumstances.

LIVING PATHOLOGY[1]

Living pathology is the important contribution of modern surgery to the medical sciences. It is the study of surgical diseases in their various stages of activity, either a return to the normal or the destruction of life, in contrast with dead pathology, which is based on the study of terminal tissue changes after the somatic death of the individual. This study of living pathology, especially in surgical diseases of the abdominal viscera, has banished old misconceptions of their clinical expression, and has given to present-day diagnosis a refinement which existed only in the dreams of a former period. It has been truly said:

> 'Tis man's worst fault to let the things
> That have been run to waste, and in the
> Unmeaning present sink the past.

The understanding which gives a meaning to present-day surgery has been developed from a knowledge of the faltering steps of scientific progress up to the creation of living pathology. Surgical history may be arbitrarily divided into three epochs. From the dawn of time to the eighteenth century, this ancient art was plied in complete ignorance of the natural processes of disease. While the acumen of observation of our forbears excites our admiration, it does not blind us to the crudity of their empiricism. With the advent of that wonderful transition era, from the darkness of medieval social, economic and moral institutions to the light of modernism, came the emancipation of medicine from the routine and thraldom of authority. To Morgagni, who introduced anatomic and pathologic thinking into medicine, must be accredited honors similar to those accorded the leaders in statesmanship, art, literature, and the culture of a marvelous Renaissance. He marks the beginning of the second epoch, the age of dead pathology. The impetus given to clinical medicine by the introduction of pathologic study, together with the continuation of experimental research, begun by Galen, to find perfection in Harvey's illustrious work, was such that medicine sought separation from art, its ancient mistress, and became espoused to science, a union that represents the turning point in the development of medicine. Its crowning glory rests with that child of a philosophic mind which the present has christened the living pathology.

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[1] Read before the Tri-State Medical Association, Madison, Wis., August, 1918.

The father of this infant prodigy, which in a few years grew to dominate surgical thought, is Lawson Tait. Observation of diseased processes through the open laparotomy wound enabled Tait to solve the riddle of the so-called broad ligament hematomas, which he discovered were due to extra-uterine pregnancy. In like manner he showed that the numerous infections of the female pelvis were, in the vast majority of instances, secondary to infection of the fallopian tubes, thus supplying the key to what had previously been known only as pelvic cellulitis. As a natural result of Tait's observations it was found that many of these disease processes could be arrested in their trend toward terminal or dead pathology.

John Hunter was undoubtedly the giant intellect of the age of dead pathology, while Lawson Tait merits the honor of having given place to living pathology as the foundation of rational surgery. This contribution, together with anesthesia and asepsis, is the Promethean gift of all time to clinical surgery. The leaders in medical science, as in the other sciences, have added luster to their age, not only by the elaboration of details, but by the evolution of far-reaching principles, destined to guide practice in many instances through centuries of darkness. This is evident when we recall that ancient medicine was based entirely on philosophic reasoning with its inherent fallacies. With the introduction of experimental medicine and the awakening appreciation of the relative rôles of cause and effect in disease, medicine emerged from behind the dark veil of superstition. For almost two centuries the practice of surgery has been based on knowledge gleaned from the study of clinical symptoms and dead pathology, and the valuable information thus gained is inestimable. Perhaps two centuries more will elapse before the medical body politic is purged of the false teachings, unavoidable in an age so pregnant with colossal truths, but this can neither cast opprobrium upon nor dim the luster of an era so wonderful in scientific progress as ours.

When Lister opened the doors of surgical possibility with the golden key of Pasteur's discovery, the new science of living pathology awaited but the master touch of Tait to make it our common possession. Only a few years have passed since then, and "the inaccuracy of thought which may come from a too exclusive devotion to the pathology of the dead" has been displaced in the minds of many by an accuracy born of attention to the pathology of the living. The progressive surgeon of today indulges the hope of that millennium when the symptoms of abdominal disease will create in the mind of every practitioner of medicine the image of living pathology. When that time comes, the phrase of the modern internist may perhaps survive, but only as a curiosity of a dead medical language; the gastro-enterologist, instead of wasting valuable energy and time in the creation of a meaningless terminology, will hasten to the side of the operating surgeon to learn the fundamental truths of living pathologic processes. Teachers of medicine will not indulge in word-pictures painted in the colors of self-delusion; endowed chairs of internal medicine will demand of the incumbent attendance at the side of the surgeon to observe and confirm the primary pathologic processes in the living. Those of you who come face to face with the difficult problems of abdominal diagnosis will bear a lighter burden if you remember the advice of Moynihan when he says: "I would urge on all those engaged in the practice of medicine the desirability of following their patients to the operating-table whenever opportunity occurs. The lessons there to be learned will in practice be of a value beyond all reckoning, and interest in the daily work will thereby be quickened to an unaccustomed degree."

Let us consider the development of knowledge relative to the living pathology of that most frequently diseased abdominal structure, the appendix. The suggestion of Grisolle, in 1837, to open and evacuate abscesses pointing anteriorly in the right iliac fossa, was made in ignorance of the origin of the pus. For centuries the pathology of the dead-house has failed to connect the wide-spread intra-abdominal lesions, to which death is due in appendicitis, with their origin in infection proceeding from that organ, so insignificant in size and so big in anatomic and physiologic importance.

To the acute mind of Reginald Fitz we owe the arguments which turned the blazing light of living pathology on the right iliac fossa, thus dispelling the mystery and revealing the truth. The cecum was cleared of the odium attached to it by the term typhlitis and perityphlitis. The results of early operation demonstrated the appendix to be the starting-point of the inflammation of that region. The bacterial origin of the disease was substantiated. The march of infection in all its stages could be seen and understood. Ulceration, gangrene and perforation of the organ and the involvement of the adjacent peritoneum with spreading peritonitis, or the formation of adhesions with the limitation of the infection and the production of periappendicular abscess—all these processes were easily understood by the opportunity afforded to observe active disease processes, which is living pathology.

Not only were the acute conditions resulting from disease of the appendix cleared up, but the frequency and importance of chronic disease became evident. It is now known that the appendix may be the seat of disease without causing symptoms of sufficient moment to excite attention. It is not at all infrequent to find an appendix to have become thickened, scarred or completely obliterated without any knowledge on the part of the patient that he is harboring a diseased organ. We also now know that a diseased appendix may express itself through some other mouth-piece and become confused with organic disease of the stomach or the duodenum or the intestines. By reflex action it may cause spastic contractions of the pylorus, pylorospasm, giving rise to symptoms purely gastric in character; or it may affect the secretion and cause the so-called secretory neuroses. Again the peristalsis and functions of the large or small bowel may be disturbed, also by reflex influences, giving rise to decreased or deficient peristalsis, with constipation, flatulence or diarrhea—so-called intestinal indigestion. This appendicular dyspepsia has been well, though incompletely, defined by Mansell as "a group of symptoms and perhaps signs which point so strongly to organic disease of the stomach or duodenum that it is only by a most careful history or by supervention of definite appendicular symptoms that a correct diagnosis is possible."

Furthermore, disease of other viscera of the abdomen are now with increasing frequency being connected with the functional disturbances due to the presence of chronic appendicitis or to a chronic bacteremia or toxemia induced thereby. It is more than a suspicion that gastric and duodenal ulcer, and inflammatory diseases of the biliary and pancreatic ducts, may often owe their origin to a chronically diseased appendix. Tubal disease and sterility of the female also has been shown to be a consequence of appendicular disease in many cases. Although today the advantage of early operative treatment of acute appendicitis is quite generally recognized, we still frequently enough see the results of neglect in acute appendicitis, which, despite a universal knowledge of the disorder, enables it to retain its distinctive "captaincy in the army of death." In acute lesions of the abdominal viscera, however, opportunities to observe the wide-spread effects of the disease are rare in comparison with our experience of thirty or even twenty years ago. But the reverse is true with regard to chronic inflammatory processes and the less frequent neoplastic diseases of the appendix.

Rarely a day passes without bringing the surgeon the opportunity, by the operative demonstration of a chronically diseased appendix, to dispel the delusion of a subacute gastric or some fanciful upper abdominal neurosis. These are mistakes of interpretation of the language of the living pathology—mistakes which, I regret to say, frequently condemn the sufferer to chronic invalidism or write his epitaph as a martyr to medical procrastination.

The practical value of a knowledge of living pathology lies not alone in increased accuracy of diagnosis, but also in the recognition of certain limitations of diagnostic possibility. It is beyond the power of man to differentiate, in certain instances, chronic disease of the gall-bladder, stomach, duodenum, pancreas, and appendix. Living pathology has taught us that disease of one or the other of these organs may, and usually does, destroy their harmony of function, and that clinical symptoms are but discordant notes common to disease processes in all. The gastric symptoms of chronic pancreatic lymphangitis, cholecystic and appendicular disease express their effects on the gastric mucosa by the action of toxins reabsorbed from the diseased area by functional disturbances depending on a common nerve supply. So rarely do we fail to find an organic basis for these digestive troubles that dyspepsia as a clinical entity has practically passed into the oblivion of surgical antiquity. It is true that disturbances in the sensory, secretory, and motor functions are not infrequently dependent on systemic disease or more rarely on defects in the mechanism of nervous control, but the entire digestive tract must be studied carefully and systematically before gastric symptoms can be attributed to these causes and be termed "idiopathic," a term which, in the great majority of cases, indicates our inability to interpret the living pathology.

I have sometimes been misled to open the abdomen in the presence of predominating gastric symptoms, only to find the pyloric sphincter to be the seat of spasm of toxic or reflex appendical irritation, as evidenced by the complete relief of symptoms following appendectomy. And on more than one occasion I have opened the stomach in search of the local cause of postprandial pain, hyperchlorhydria, or pylorospasm, to find disappointment in the presence of a perfectly healthy mucosa. Even hematemesis has been disproved as a symptom of simple ulceration in a number of instances, and found to depend on the toxic effect on the arterioles of the gastric circulation, or of poisons generated in a distant and diseased organ with the production of simple erosion of the mucous membrane, as described by Dieulafoy. Chronic simple ulcer of the stomach and duodenum is believed by Mayo Robson and others to result from oral and other septic processes, and the frequent association of chronic appendicitis with duodenal ulcer has been explained in the same manner.

It is not my purpose to discourage attempts at differential diagnosis, for this is of the utmost importance and, as a rule, possible; but more intelligent by far is the physician who recognizes the voice common to the living pathology of various structures, than he who attempts to localize the trouble to a particular organ, and then attributes it to functional derangement.

Let us consider the influence of our knowledge of the living pathology of the pancreas and biliary passages on the modern conception of their diseases. Bacteriologic investigation of the contents of the diseased gall-bladder has clarified our ideas of the origin of cholecystic disease, and the living pathologist traces, in the order of their sequence, the steps in the progress of the disease from the reception of the micro-organisms through the stages of acute and chronic inflammation and their complications, gall-stone formation, precancerous tissue changes and, finally, carcinoma. In 1896 Riedel, in operating in a case of gall-stone disease, discovered a chronic pancreatitis, which he differentiated from primary cancer of the head of the organ. Since that time the opportunity for observation which has been afforded by surgery has established the fact that the pancreas is frequently the seat of chronic inflammatory alterations. The touch of the surgeon during life has revealed that which postmortem pathology had overlooked. It has also been possible to trace the path of infection from the gall-bladder through the lymphatics lying in the right free border of the gastrohepatic omentum to the peripancreatic plexus. The lymph-nodes clustering about the pancreas are seen to be enlarged, and from their anastomosis with the intrapancreatic lymphatics the assumption is fair that the pancreas is not infrequently infected in this manner. To this early inflammatory disease of the organ the name pancreatic lymphangitis has been applied. That it is a precursor of the more serious and pronounced changes in the pancreas there can be no doubt.

This fact of living pathology has supplied us with the key for treatment of this form of pancreatitis through drainage of the biliary tract. When the source of the lymphatic infection is thus removed, recovery of the pancreas is the rule, as has been demonstrated on occasions when subsequent operation became necessary for some other reason, as well as by the subsidence of symptoms after operation.

The early hope of the surgical relief of diabetes, when this condition is secondary to chronic inflammatory processes in the pancreas, has not been fulfilled. Destruction of the islands of Langerhans by cicatricial contraction of inflammatory tissue bespeaks a process too far advanced for relief by drainage. It denotes a parainfectious condition, the terminal change in the surgical conception of a process that probably would have been amenable to operation at an earlier stage, had the physician in attendance heard in the digestive complaints, not the voice of functional disturbance, but an echo of the cry of distant organic disease.

Obstinate constipation is another field formerly considered exclusively medical and beset with unfounded theories, which are now being subjected to the search of the living pathologist. Sir Arbuthnot Lane, who through active work, has described with great plausibility certain mechanical factors in the form of bands and kinks which embarrass peristalsis and give rise to intestinal stasis. Whether or not we are willing to go all the way with him in his ideas, we must acknowledge that there is truth at the bottom of the well and that it will in the end rise and prevail as a result of demonstrations on the living subject.

That duodenal ulcer is today so frequently observed is exclusively due to the necropsy *in vivo*. Only a few decades ago ulcer of the duodenum was considered a rare lesion and its symptoms were parading under many painful gastro-enterologic names, such as hyperchlorhydria, gastralgia, chronic gastritis, etc. Gastric ulcer was thought to be far more common than ulcer of the duodenum. Today the order of frequency is reversed, and a typical syndrome of symptoms has been worked out by means of which the more typical cases may be diagnosed with great accuracy. Still there are many ulcers which give but little evidence of their presence and it is easy to overlook the lesion.

If further illustrations were necessary to prove our contention of the radical difference between living and dead pathology, we need but to contrast the postmortem records of any large hospital with the clinical statistics based on operative findings. Search the records of any pathologic laboratory, and you will rarely find an instance of peritonitis secondary to a perforation of a chronic calloused ulcer of the duodenum. Yet it has been my privilege to operate on a large number of patients during the past ten years with this alarming complication of ulcer, and I have had 98 per cent. recoveries.

As another example, I will mention that instances of death from calculous obstruction of the common bile-duct are rare in hospital records. Would not clinical teaching based on records be highly erroneous? The practical significance of the matter is that numerous patients, as a result of these conditions, die outside of the hospital, and that the few are saved by following the advice of an intelligent physician who, cognizant of the living pathology, realizes the necessity of immediate surgical consultation. Furthermore, bacteriologic study, in cases of ruptured gastric and duodenal ulcers, has taught us that during the first twelve hours the peritoneal fluid frequently is sterile, and in this we find the reason for the almost perfect success of early operative interference. This early sterility of the extravasated fluid enables us not only to undertake closure of the perforation, but also plication of the duodenum and posterior gastro-enterostomy as well, procedures that in the great majority of cases relieve the patient of future ulcer trouble and offer an assurance of operative success almost equal to the certainty of death when, after the lapse of a few hours, the peritoneum has become infected. The profound predisposition to cancer of a chronically ulcerated area of the pyloric antrum is one of the startling facts exposed to the light of day by the aseptic scalpel. Knowledge of the cellular pathology of neoplastic tissue removed at operation has completely rearranged our ideas of cancerous change, and forms the basis of the only rational treatment—early and radical excision of ulcers. The microscopic demonstration of the very earliest malignant changes in the bases of chronic ulcers of the stomach in association with precancerous inflammatory tissue has added the necessity of excision of areas of potential carcinoma. Indeed, this relationship has been so definitely proved that it is the aim of every surgeon whose judgment is based on living pathology to prevent the necessity of radical operations on the uterus, stomach, breast, and other organs by the early recognition of malignant degeneration of primary benign tissue changes.

May I venture to call attention to another of the contributions of living pathology toward the saving of human life? This relates to our present knowledge of renal tuberculosis. Formerly, when death occurred from tuberculosis of the kidney, it was the rule for advanced tuberculous processes to be found elsewhere. Very frequently both kidneys were involved and, as a rule, the bladder showed extensive tuberculous ulceration. By the study of renal tuberculosis in motion several important facts have become apparent: First, that tuberculosis of the bladder is never primary, but always secondary to disease of the upper urinary tract or of the genital tract, and that it most frequently follows infection of the kidney. Secondly, it has been found that in the beginning surgical tuberculosis of the kidney is usually unilateral. Disregarding the abstract question of whether the infection is distinctly primary or secondary to tuberculosis of some other portion of the body, it is true, at least, that the renal lesion may be the only active focus of tuberculosis in the body. This knowledge has led to nephrectomy for renal tuberculosis, by which many lives have been saved.

We could multiply examples in the citation of various surgical diseases of the abdominal viscera to illustrate the contributions of living pathology to a clearer understanding of conditions which so frequently manifest themselves through the medium of gastric symptoms, but I trust enough has been said to present a glimpse of the pathway of progress.

Only by standing at the elbow of the surgeon, let me repeat, can the internist hope to attain proficiency in diagnosis and the proper conception of treatment in these conditions. Observe that I say the elbow of the *surgeon*, not the operator, for there is a sharp distinction between the mechanical practitioner of surgery and the true surgeon, who is a specialist, not in operating, but in the field of surgical disease, skilled in diagnosis and prognosis, aware of the dangers and limitations of his art, and as ready to withhold his hand at the bidding of science and conscience as he is bold to attack the condition which, unchecked, threatens the patient's welfare.

"A duty," says the poet, "is a demand of the hour." The duty of the general practitioner is recognition of the advantages of studying the living pathology and a willingness to employ every means to this attainment. To those whose symptomatic treatment of surgical disease is gilded with the self-delusion of rationality, I would commend that sublime utterance of Polonius:

> This above all: to thine own self be true,
> And it must follow, as the night the day,
> Thou canst not then be false to any man.

INDEX

Abdomen, "acute," 121
        purgation in, 121
Abercrombie, 2
Alonzo, 123
Anemia, pernicious, 62
        differential diagnosis, 62
Anesthesia, choice of, 111
    discovery of, 153
Anesthetist, nurse, 111
Antisepsis, birth of, 155
Appendicitis, 117
    acute, 118
        leukocytosis in, 121
        operation, choice of, 123
        pain in, 120
        symptoms of, 120, 142
        treatment, postoperative, 124
        vomiting in, 120
    and gastric ulcer, 35
    chronic, abdominal symptoms of, 174
        and sterility, 174
        and tubal disease, 174
        causes of, 174
        definition of, 141
Appendix, acute, 173
    chronic, 173
    carcinoma of, 143
    positions of, 118
    varieties of, 142
Aschoff, 72
Asepsis, development of, 155
Atlee, 102

Bacmeister, 72
Bacillus, Boas-Oppler, 137
    paratyphosus, 94
    tubercle, 133
    typhosus, 94
Baillie, 1
Balfour, 16, 21
Bamberger, 27
Banti's disease, 61, 66
Batzdorf, 144
Bernard, Claude, 29
Bile, bacteriolytic action of, 95
    passage of, to gall-bladder, 86
    storage of, 85
Bile-ducts, inflammation of, 70
Bile-passages, 67
    infection of, 67
        route of, 67
Blood transfusion after splenectomy, 65
Bolton, 4, 14
Bowel, large, points of delay in, 127
        ulcer of, 125
Braun, 2
Brown, 94
Bunting, 144
Burgess, 120

Calculi, biliary, formation of, 71
Cannon, 39
Carcinoma, cecum, 129
    colon, 127
    diagnosis, x-ray, 129
    duodenum, 14
    gall-bladder, 99
    gastric, history of, 48
        relation to ulcer, 45
    intestines, treatment, surgical, 131
    rectum, infection, secondary, 131
        obstruction of, 130
        prognosis, 132
Carlson, 90
Carter, 127
Chambrun, 151
Chauffard's disease, 60
Chiropractors, 166
Cholangitis, 72
Cholecystitis, 72
    acute, operation, choice of, 80
            time for, 79

Cholecystitis and hepatitis, 69
  calculous, 75
    classification of, 75, 77
    diagnosis, *x*-ray, 78
    treatment, 74
      palliative, 78
      surgical, 78
  gangrenous, 19
  non-calculous, 74
  pathology of, 84
  recurrence of symptoms, 80
  relationship to pancreatitis, 81
Cirrhosis, liver, splenectomy for, 64
Clark, 123
Codvilla, 2
Colic, gall-stone, 20
Common bile-duct, obstruction of, 54
    calculous, 55, 179
Constriction, hour-glass, of stomach, 13
Co-operation, surgeon, internist, laboratory, 115
Corbin, 133
Crile, 2, 123
Cruveilhier, 1
Czerny, 2

Deaver, 34, 37, 38, 45, 90, 92, 95, 97, 98, 141, 145
Diabetes, 178
Diagnosis, accuracy in, 175
  differential, 175
  medical, 105
  surgical, 108, 109
Dieulafoy, 76
Diverticulitis, 125
Doyen, 2
Dyspepsia, appendicular, 174

Education, medical, 161
Eppinger, 54, 59
Erdman, 127
Erosion, 3
Ewing, 144
Experimentation, animal, 114

Fermentation, germ-life in, 150
Fever, puerperal, 157
Finney, 12
Fitz, Reginald Heber, 118, 173
Follow-up records, 113
Fowler, 123
Frank, Johann Peter, 1

Galen, 170
Gall-bladder, bacteriology of, 68
  carcinoma of, 99
  connective-tissue changes in, 91
  contractions of, 88
  disease of, pathology, 84
  drainage of, non-surgical, 74
  functions of, 84
    concentrating, 84
    propulsive, 87
  infection of liver cells in, 69
      route of, 82
      organisms in, 68
        pneumococci, 94
        streptococci, 95
  inflammation of, 70
    interstitial, 92
    pathology, 90
  lymphatics of, 69, 95
  perforation of, 91
  relation to liver, 95
  tumors of, pathology, 90
Gastrectomy, partial, 13
Gastric resection, 20
Gastro-enterostomy, healing action of, 43
Gaucher's disease, 61
Graham, Ewarts, 68, 69, 73, 95
Grisolle, 172

Hallowell, 133
Hanot's disease, 53, 61
Harvey, 170
Helmbach, 71, 72
Hemorrhage, gastric, 4
Hemorrhoids and carcinoma, 126
Hepatitis, acute, 73
  and cholecystitis, 69

Hippocrates, 1
Hoffman, Friedrich, 1
Hooper, 57
Hospital, mortality statistics, 156
Hunter, John, 29, 170
Huxley, 114

Inflammation, appendical, localization of, 122
Internist, co-operation between surgeon and laboratory, 115
Internships, 166
Intestinal tract, carcinoma of, 125
        incidence of, 125
            age, 126
            site of, 125, 145
        surgical conditions of, inflammatory, 117
            malignancy, 117
            pathology of, 138–140
        tuberculosis of, 117, 133
Intestines, length of, 146

Jacobson, 75, 82
Janeway, T. C., 163
Jaundice, bile pigments in, 57
    characteristics of, 56
    itching in, 56
    stools, acholic, 56
    types of, catarrhal, 58, 73
        dissociated, 59
        hemolytic, 58, 59, 60
            relation to liver and gall-bladder, 60
        obstructive, 55
        relapsing, 73
    urine in, 56
Jejunum, ulceration of, primary, 124
Jenner, 152
Judd, 73, 81

Kehr, 73
Keith, 126
King, 57
Koch's tubercle bacillus, 133
Kraske, 126
Krause, 2
Kriege, 2
Krumbhaar, 6
Kupfer's cells, 96

Laboratory, co-operation between surgeon and internist, 115
Lane, Sir Arbuthnot, 178
Law of localization, 5
Lee, 79
Linder, 81
Lister, 103, 104, 115, 138, 153, 155, 156, 171
Living pathology, 97
Long, 138
Lubarsch, 144
Luschka's glands, 92

Mallock, Archibald, 2
Mansell, 174
Mayo, C. H., 3
Mayo, W. J., 3
Mayo-Robson, 2
Mayos, 2, 82
McBurney's point, 120
McDowell, 102
McMaster, 71, 84, 85
Meckel's diverticulum, 125
Medical school, function of, 164
Medicine, Academy of, Paris, 157
Microbes, transmissible, 152
Mikulicz, 2
Morton, 138
Moynihan, Sir Berkeley, 2, 3, 4, 7, 10, 14, 110, 133, 172
Mucosa, gastric erosion of, 3
Murphy, John B., 118, 123

Naunyn, 71
Neurosis, gastric, 9

Ochsner, 113
Oddi, 84
    sphincter of, 74
Operation, ritual of, 110
Operations, Balfour, 16
    Kraske, 126

Operations, Mikulicz, 128
  Polya, 13
  Roux-y, 24
Organisms, elective affinity of, 4

PAGET, Sir Stephen, 158
Pancreatic lymphangitis, 83
  plexus, 177
Pancreatitis, acute, 19
    hemorrhagic, 98
  treatment of, 177
Papilla of Vater, 15
    anatomy of, 98
    obstruction of, 54
Paré, Ambroise, 116
Pasteur, 103, 114, 138, 149, 150, 151, 152, 155, 156, 157, 158, 171
Pathology, cellular, 180
  living, 169
    development of, 172
Peritoneal fluid, early sterility of, 179
Peritoneum, resorptive power of, 121
Peritonitis, 124–173
  treatment of, ideal, 122
Peterman, 69, 95
Polonius, 183
Polya operation, 13
Practical men, need for, 162
Professorship, full time, 163
  pros and cons, 166
Pylorectomy, 13, 17, 20
Pyloroplasty, 12
Pylorospasm, 9
Pylorus, identification of, 3

RADIUM, 51
Ravdin, 14
Raynaud's disease, 3
Rectum, carcinoma, 130
Reeves, 15
Reimann, 69
Ribbert, 134
Robson, Mayo, 176
Rokitansky, 27
Rolleston, 68
Rosenow, 4, 32, 93, 94
Rost, 59, 72
Rous, 71, 84, 85
Ruggi, 146
Rydygier, 2

SCIENCE, American, position of, 107
Scleroderma, 3
Shaw, 2
Sippy, 10
Spleen, enlargement of, congenital, 61
  weight of, 63
Splenectomy, blood transfusion in, 65
  end-results, 66
  technic of, 64
Splenomegaly, 60
Sterility due to appendicitis, 114
Stewart, 57
Stomach, chemistry of, after gastroenterostomy, 42
  physiology of, 38, 39
Stiles, Sir Harold, 106, 107
Stone in hepatic duct, 81
Sudler, 69
Surgeon and internist, co-operation of, 115
  clinical work of, 105
  modern, 104
  responsibilities of, 108
  training of, 106
  trials, tribulations, and joys of, 101
  tribulations of, 112, 114
  Surgeons, American College of, 106
  barber, 103
Surgery, co-operative thought in, 107
  empiricism in, 101
  epochs of, 169
  follow-up records in, 113
  former technic of, 154
  modern, Pasteur's contribution to, 149
  technic of, modern, 156
  triumphs of, 104
Surgical diagnosis, 108
Symptoms, abdominal, caused by appendicitis, 174
Syphilis of liver, 58

Tait, Lawson, 102, 170
Tests, laboratory, usefulness of, 110
Tubercle bacillus, Koch's, 133
Tuberculosis, intestinal, 133
    associated lesions of, 134
    diagnosis of, 136
        differential, 136
    dissemination of, 135
    etiology of, 134
    sites of, 135
    symptoms of, 135
    treatment of, surgical, 137
    types of, 134
  renal, 180
    pathology of, 181
Tumor, definition of, 46
Typhoid fever, 68, 69

Ulcer, cecum, 125
  duodenal, 2, 14, 178
    acute perforating, 18
    characteristics of, 15
      hematemesis, 15
      hunger pain, 15
      jaundice, 15
      latency, 15
      melena, 15
      periodicity, 15
      vomiting, 15
    diagnosis, 16
      differential, 16
    incidence, 14
      sex, 14
    location, 14
    multiple, 14
    perforating, 2, 179
      treatment, gastro-enterostomy, 20
        end-results, 22
        surgical technic, 22
    treatment, surgical, 16
      excision, 17
  gastric, 2, 4, 6
    acute, perforating, 18
    carcinomatous transformation of, 11
Ulcer, gastric, carcinomatous, transformation of, incidence, 49
    characteristics of, 6
      fever, 7
      hematemesis, 7
      hemorrhage, 7, 9
      vomiting, 7
    classification of, 4
      round, 5
      saddle back, 13
    diagnosis of, 8
      acidity, 9
      differential, 9
      gastric analysis, 8
      pain, relief of, 8
      x-ray, 8
    etiology, 33
      appendicitis, 35
      experimental, 29
      nervous, 33
      sex, 6
    incidence, autopsy, 35
    secondary anemia in, 7
    treatment, medical, 10
      surgical, 11
        end-results, 13
        excision, 11
        posterior gastro-enterostomy 11
            effects of, 36
        pylorectomy, 11
        subtotal gastrectomy, 11
  intestinal, 125
  marginal, 22, 34
    diagnosis, 23
      x-ray, 23
    etiology, 22, 23
      perforating, 24
    symptoms, 23
    treatment, surgical, 24
  peptic, 1
    bacteriology, 32
    cancerous change in, 180
    history, 1
    incidence, 18, 37
    location of, 28

Ulcer, peptic, pathology, 27
    perforating, 17
      acute, diagnosis, 19
      incidence, 18
    relation to carcinoma, 45
    secondary disease, 34
    treatment, effects of, 40, 41
        chemical, 42
      postoperative, 24
      surgical lavage, 26
  sigmoid flexure, 125
  typhoid, 124
    sites of, 125
Ulcus ventriculi, 2
Urine, discoloration of, in jaundice, 56

VACCINATION, 152
Vein, pyloric, 3
Vincent, 79
Virchow, 27
Vomiting, 15

WEIL's disease, 58
Weir, Robert, 2
Whipple, 57, 79
Wilkie, 15
Wound infection, 153